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REVIEW ARTICLE

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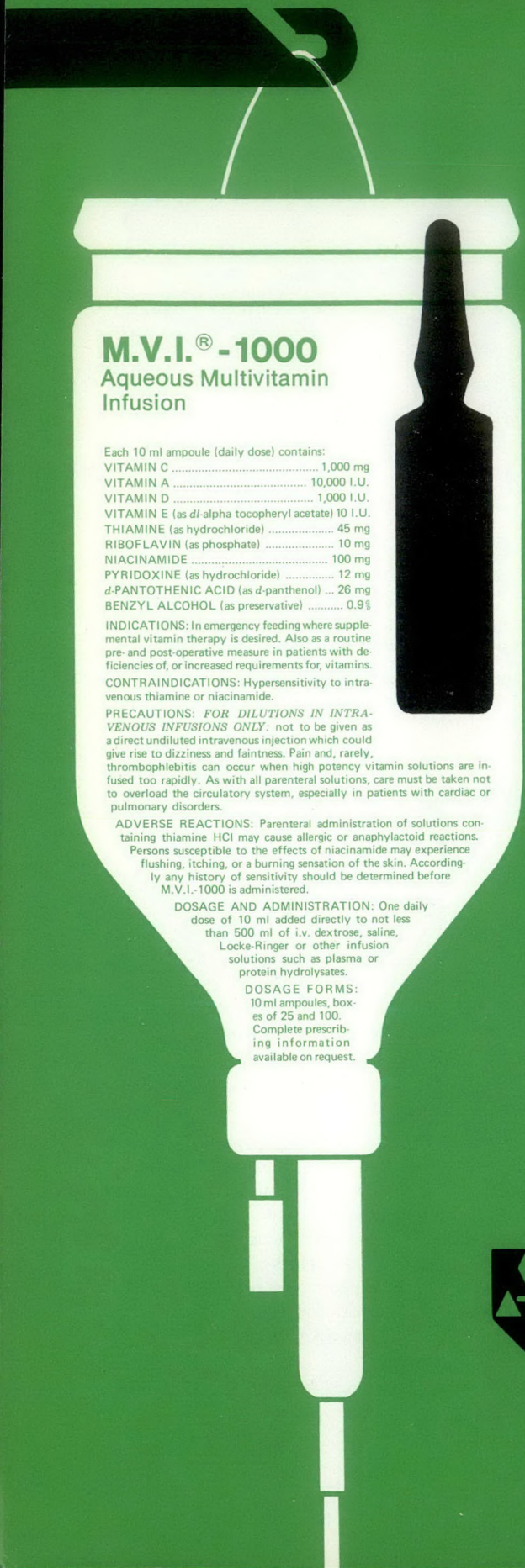

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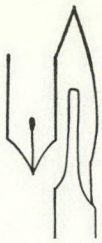

QUILL ON SCALPEL This section provides a medium through which Canadian surgeons can declare themselves, briefly and informally, on the day-to-day affairs of surgery.

THE LOWER ESOPHAGEAL SPHINCTER AND REFLUX ESOPHAGITIS

Reflux esophagitis, with or without stricture, has been treated successfully by many different operations. Gastric resection, with a decrease in acid production and prompt gastric emptying, seemed logical because many patients with the severest form of esophagitis (i.e. stricture) have duodenal ulcer and increased acid production. However, this operation may itself be followed by esophagitis in a patient in whom this never occurred before gastrectomy. Bile has been implicated as the cause in this clinical setting. This conclusion is supported by generally good clinical responses when bile is diverted away from the gastric remnant.

Currently, therapy for reflux esophagitis has been directed at restoring a "normal" lower esophageal sphincter, it being recognized that most, if not all, esophageal strictures that result from reflux can be dilated. This approach has been strongly supported by pressure measurements in the stomach and esophagus before and after operation.

In patients with reflux esophagitis the stomach and esophagus are a common cavity and when external compression is applied to the abdominal wall the pressures measured simultaneously in the stomach and esophagus are frequently equal. Operations that reposition the esophagogastric junction within the abdomen can restore a "high pressure zone" in the distal esophagus, restore competence and thereby eliminate the common cavity. It is generally agreed that a physiologic sphincter that responds appropriately to abdominal compression to prevent reflux is created by the Hill, Belsey or Nissen operations. A good clinical result can follow an operation that includes no attempt at crural repair of esophageal hiatus.

Bowes and Sarna, in this issue of the Journal (page 328), in a study of patients after a modified Nissen operation, emphasize the mechanical effects of these operations. Narrowing of the esophagus in the operative area increases the resistance to flow, which is what a perfused catheter system is measuring, and this is recorded as a high pressure zone. These workers did not detect active relaxation during appropriate stimulation. While the "sphincter pressure" increased with abdominal compression, the ratio of change in sphincter pressure to change in gastric pressure was not different after operation, which suggests a mechanical effect. *In-vitro* studies on cadavers demonstrated that a zone of elevated pressure could be produced by extrinsic compression of as little as 50 g over a 3-cm length of esophagus.

Finally, Bowes and Sarna observed patients whose symptoms were relieved and in whom an increase in sphincter pressure did not occur; and one patient who developed recurrence despite significant increase in sphincter pressure.

These studies are important and suggest that a high pressure zone, when it is created surgically, is mechanical and does not resemble a physiologic sphincter. This might explain why a "high pressure zone" can occur in colon brought through the diaphragm.

Surprisingly, an increase in sphincter pressure is not necessary for creation of a competent gastroesophageal sphincter. Further studies should reveal the mechanism that creates gastroesophageal competence and thereby might clarify the requirements of an operation for gastroesophageal reflux.

L. D. MacLean, MD, FRCS[C]

REVIEW ARTICLE

CLINICAL USEFULNESS OF GASTRIC ACIDITY STUDIES*

J. E. MULLENS, MD, MS, FRCS[C], FACS

Summary: Tests of gastric secretion are seldom diagnostic in the investigation of gastric and duodenal ulceration, but they can provide evidence that is helpful in arriving at a diagnosis. Common tests include basal acid output, pentagastrin-stimulated maximal and peak acid output, and the Hollander test. There is some evidence that they may be useful in selecting the type of operation for peptic ulceration. Indications for performing these tests relate to the Zollinger-Ellison syndrome, duodenal ulcer, recurrent dyspepsia after operation for duodenal ulcer, and decisions concerning the choice of operation for peptic ulcer. In order to perform these functions they must be properly conducted and interpreted; they are simply an adjunct to clinical judgement, and complementary to other laboratory tests.

Résumé: Les épreuves de la sécrétion gastrique n'ont guère de valeur dans le diagnostic des ulcères gastrique et duodénal, mais elles peuvent constituer des adjuvants utiles pour y arriver. Les épreuves les plus courantes sont le débit acide fondamental, le débit acide maximal et le débit acide de pointe après stimulation par la pentagastrine et l'épreuve de Hollander. On a des raisons de croire qu'elles peuvent être utiles pour le choix du type d'opération d'un ulcère gastro-duodénal. Les indications justifiant ces épreuves sont le syndrome Zollinger-Ellison, l'ulcère duodénal, la dyspepsie secondaire à un ulcère opéré et facilitent à prendre les décisions concernant le choix de l'opération à pratiquer dans un ulcère gastro-duodénal. Pour qu'elles remplissent ces fonctions, les épreuves doivent être pratiquées et interprétées judicieusement. Elle ne sont que de simples adjuvants du jugement clinique et ne font que compléter les autres épreuves de laboratoire.

TESTS of gastric acid output, when properly conducted and interpreted, contribute useful information to the diagnosis and management of peptic ulceration. They do not give information that is any more conclusive or pathognomonic than that given by any other of the biochemical and physiologic tests conducted on patients with disease of the gastrointestinal tract, and perhaps because too much is expected of the tests some clinicians are sceptical about their use. Use of these tests is discussed in several excellent reviews;[1-7] this paper is a summary of their clinical application.

COMMON TESTS OF GASTRIC SECRETION

Three tests of gastric secretion are commonly used: (a) the basal acid output (BAO); (b) pentagastrin stimulation, including, first, maximal acid output (MAO) and, second, peak acid output (PAO); and (c) the Hollander test. Additional tests that may be used under special circumstances include the glucagon or secretin test and the calcium infusion test. All tests should be conducted by a nurse or technician specially trained in the procedure, and scrupulous attention to detail is essential if they are to provide reliable information. Particularly helpful is a laboratory with staff having a special interest in the tests and who know what is expected of them. Repeated consultations with the biochemist are required to maintain a consistent quality of reporting.

Basal Acid Output

The acid secreted from the stomach in the interdigestive phase or basal phase ranges from 1 meq/h to 3.0 meq/h in the normal subject. Caucasian women secrete at an average rate of 1.5 meq/h; for men the rate is 2.5 meq/h. In persons of other races there are different values for normal for both the BAO and MAO.[8, 9] Such racial differences are probably attributable to differences in the average age, sex and body weight in the subjects that have been examined.

The basal specimen may be collected over any period when basal conditions exist. A 12-hour overnight collection has initially been used to provide this specimen, but it is difficult to ensure basal conditions and accurate collections over this period, and

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there is now no apparent advantage over the 1-hour basal collection. The latter is probably more reliable, and it is easier for the patient and staff.

Stimulated Gastric Acid Output

Maximal acid output is the total output of acid over a 1-hour period expressed in milliequivalents per hour. The true maximal capacity of the stomach to secrete is not measured, but the determination of "maximal acid output" is sufficiently close to the true maximal capacity of the stomach to secrete and it is reliably reproducible, so that the value for MAO is quite satisfactory for clinical purposes.

The PAO is defined as the amount of acid secreted in the highest values for any two 15-minute periods of a 1-hour test, added together and multiplied by 2. This gives a closer determination of the maximal capacity of the stomach to secrete acid in a 1-hour period. The PAO is replacing the MAO as an expression of the acid-secreting capacity of the stomach, but the values for the two tests are usually very close together and almost interchangeable.

Pentagastrin has supplanted all other types of stimuli in inducing gastric secretion for the measurement of MAO and PAO.

Hollander Test

Hypoglycemia is a stimulus to acid secretion and, for practical clinical purposes, the stimulus of hypoglycemia is mediated entirely through the vagus nerve; that is, if the vagus nerves are not intact hypoglycemia will not increase the gastric acid output, at least in the 1st hour after the stimulus.

The only function of the test is to determine whether any portion of the vagus nerves remains after vagotomy; therefore, the test should be performed only in patients who are being investigated for persistent or recurrent symptoms after vagotomy. It was once thought that the response to stimulation of the vagus nerve was an all-or-none phenomenon, but it is now realized that there are graded responses to hypoglycemia, depending on the number of vagus fibres that remain intact.

In the Hollander test, insulin is administered to provoke hypoglycemia. Other agents (tolbutamide and 2-deoxy-D-glucose) have been used to replace insulin but these agents have no advantages over insulin, and possibly some disadvantages. The response of the stomach to hypoglycemia is the secretion of hydrochloric acid (measured in milliequivalents of acid). If no vagal fibres remain, there should be no increase in the output of acid over the basal levels.

Method of Performing the Tests

Basal, Maximal and Peak Acid Output

A radiopaque nasogastric tube is passed into the stomach. The tube is preferably of a sump type, such as the Salem tube, which is less likely to become blocked and require irrigation. The tip of the tube is positioned in the body of the stomach under radiographic control. The stomach is emptied and that specimen is discarded. Then, while the patient remains in a left semilateral position, the secretions of the stomach are collected over the next hour by constant suction. This first specimen is labelled "basal acid output".

An injection of pentagastrin (6 mg/kg) is given subcutaneously. Collections are made by constant suction over a 1-hour period and kept separate as four 15-minute specimens. These are labelled as four separate specimens and sent with the BAO specimen to the laboratory. The laboratory should report these five separate determinations in terms of milliequivalents per hour of acid titrated to neutrality. Reports in terms of "free acid" (i.e., to an end point of pH 3.5) should no longer be used.

A dye-dilution indicator may be given to determine the amount of gastric juice lost through the pylorus, but there is little need for this in the clinical tests of gastric secretion, nor is it necessary to determine the amount of bile in the specimens. It is the net acidity that is measured. This figure is reproducible in the same subject on repeated testing. The interpretation of results depends on a comparison with, or reference to, the acidity studies in a large group of subjects. If the results are abnormally high or abnormally low, one can make a valid comparison with the disease processes that occur in the larger group.

Hollander Test

The Hollander test[10-13] should not be used in patients with a history of heart disease, those with cardiac arrhythmias or those who are over 60 years of age. Deaths have occurred from its use.

The nasogastric tube should be passed and positioned as in the other tests. The first specimen is discarded. A basal 1-hour sample is then collected and labelled. Through an intravenous line, an infusion of isotonic saline is started. On one limb of a Y-connection, a bottle of 5% dextrose in water is set up but not started. A fasting blood sugar sample is taken when the intravenous infusion of saline is started. The optimal dose of crystalline insulin (0.2 units/kg) is then administered intravenously and four 15-minute specimens are collected by aspiration from the nasogastric tube. There is no advantage in collecting during the 2nd hour to measure the "late response"; it is now thought that the distinction between "early" and "late" responses probably has no prognostic significance.[11, 14] When the patient manifests the signs and symptoms of hypoglycemia, a blood sugar sample is taken and then the hypoglycemic reaction is terminated by infusing the dextrose solution. For a valid test the blood sugar should be less than 40 mg/dl.

Glucagon and Secretin Tests

Glucagon and secretin have been used to provoke an increase in the serum gastrin concentration in patients with the Zollinger-Ellison syndrome.[15, 16] The gastric acidity may rise in patients with this syndrome, but the object of the test is to determine if the serum gastrin value increases and is not strictly a study of gastric acidity. In patients with the Zollinger-Ellison syndrome the serum gastrin value will increase abruptly within the 1st hour after intravenous administration of glucagon (1 mg) or secretin (2 units/kg). Intractable, recurrent ulceration after operation can be a result of the Zollinger-Ellison syndrome or of retention of an excluded antrum. The secretin or glucagon test can be used to point to a diagnosis. The serum gastrin value does not increase after the administration of glucagon or secretin in a patient who has retained antrum, and the MAO usually falls.

Although the next group of tests are not strictly tests of gastric acid secretion they are carried out in a similar manner and are used in the investigation of intractable or recurrent ulceration. Therefore these tests should be considered at this point.

Calcium Infusion Test

The calcium infusion test may also be of value when the Zollinger-Ellison syndrome is suspected.[17-19] It has been recommended that the test not be done on those with cardiac or renal disease.[18] This test is done in two stages. In the first stage, calcium gluceptate* (2 mg/kg) is given intravenously. If gastric output of acid does not increase, the Zollinger-Ellison syndrome is unlikely. The second stage of the test is performed if there is an increase in acid output.

In the second stage, calcium gluceptate (15 mg/kg) is infused intravenously over 3 hours. Blood samples are taken for serum gastrin assay. If the output of gastrin during the calcium infusion test increases, the Zollinger-Ellison syndrome is likely; if there is no increase, this syndrome is unlikely. In patients with duodenal ulcer, there should be no increase in the output of gastrin. (The calcium infusion test may also be used to distinguish the Zollinger-Ellison syndrome from retained excluded antrum.)

Tubeless Tests

Use of the Heidelberg capsule with telemetry has been recommended to ascertain the capacity of the stomach to secrete acid.[20] Results of these tests using this method have not been dependably reproducible, and they are not recommended for general adoption.[4]

Indications for Tests of Gastric Secretion

Zollinger-Ellison Syndrome

When the Zollinger-Ellison syndrome is suspected, gastric acidity tests may provide information that will suggest whether gastrin

*Obtainable from Eli Lilly and Co. Ltd. or Abbott Laboratories, Ltd.

assays are necessary. Further, when the results of the secretion studies are considered in conjunction with the results of the serum gastrin assays they will be helpful in reaching a diagnosis. The following results are strongly suggestive of the presence of the Zollinger-Ellison syndrome:

- BAO: 1. Patients not operated on, $\geqslant$ 15 meq/h
  2. Patients operated on, $\geqslant$ 5 meq/h
- MAO: 1. Patients not operated on, $\geqslant$ 100 meq/h
  2. Patients operated on, $\geqslant$ 70 meq/h
- BAO/MAO: $\geqslant$ 60%
- BAO/PAO: $\geqslant$ 60%.

Duodenal Ulcer

When a patient has symptoms of duodenal ulceration, and radiography and endoscopy do not reveal an ulcer, tests of gastric secretion may be of diagnostic value. The results of gastric acid secretion testing in normal subjects of average size and weight overlap those of gastric acid secretion in duodenal ulcer, so that testing is of little value in diagnosis when the results lie within the range of this overlap. Nevertheless, there is a threshold of acid secretion below which duodenal ulcer does not occur, and there is a threshold of secretion above which duodenal ulcers almost always are found. A PAO of less then 15 meq/h is strong evidence that a duodenal ulcer does not exist, and a PAO greater than 50 meq/h is strong evidence that a duodenal ulcer does exist. Blackman and associates[8] have shown that in 90% of patients with duodenal ulceration PAO was outside the normal range and that in 92% of normal subjects PAO was within the normal range if age, sex and weight were taken into account in calculating the results of the tests. These authors have described a formula for making these calculations.

The BAO is of little use in making the diagnosis of duodenal ulcer, but a BAO exceeding 5 meq/h strongly suggests the presence of an ulcer. The BAO may also give a clue to the presence of the Zollinger-Ellison syndrome, and therefore should be determined whenever the tests of gastric secretion are performed.

Recurrent Dyspepsia after Operation for Duodenal Ulcer

If a patient complains of recurrent dyspepsia after an operation for duodenal ulcer, both the Hollander test and the pentagastrin test may be of help in determining the existence of a recurrent ulcer.

The Hollander test should be done in selected patients when a vagotomy has been performed. If the patient has had a vagotomy and the Hollander test is positive, the chances of recurrent ulcer are increased,[1, 21] although some reports indicate that the recurrence rate is almost as high in those in whom the test is positive as in those in whom it is negative.[13, 22, 23] Recurrent ulcer may develop after a complete vagotomy, but the Hollander test is a guide for further surgical management if such is the case; for example, if the vagotomy has been complete, as indicated by the Hollander test, there would be no need to reexplore the vagus nerves. The routine performance postoperatively of Hollander tests is probably of little value in assessing the risks of recurrent ulceration.[2, 7, 23] If a complete antrectomy has been performed, one cannot expect a positive Hollander response, even in the presence of retained vagal fibres.[24]

There has been much debate about the criteria of a positive Hollander response. It has been suggested that multiple criteria of a positive response are better than Hollander's original criterion, but there is no consensus on this.[21] Hollander's original criterion[12] probably is as dependable as any index of the presence or absence of intact vagal fibres; that is, an increase in titratable acidity of 20 meq/*l* in any two consecutive 15-minute samples in the 2 hours after intravenous administration of insulin (dose, 0.2 units/kg) compared with the mean acidity of two 30-minute basal samples. If the basal sample contains no "free acid" (pH $>$ 3.5) the test is positive provided the acidity after administration of insulin is equal to or exceeds 10 meq/*l*.

The values for BAO after operation are not completely dependable, but a value greater than 5 meq/h is found only in pa-

tients with recurrent ulcer. One must be aware that the basal acid output may remain high even after a complete vagotomy.[25] Moore[26] has also shown that an intravenous infusion of 5% dextrose reduces BAO. In other words, a true BAO will not be determined in any patient in whom a solution of 5% dextrose is being infused.

The pentagastrin test also offers some diagnostic information: an MAO exceeding 15 meq/h is unusual except in cases of recurrent ulcer, and an MAO exceeding 22 meq/h is found only in cases of recurrent ulcer.

Deciding the Choice of Operation for Peptic Ulceration

Several attempts have been made to use the tests as a method of choosing the appropriate operation.[5, 7, 21, 23, 27-29]

In planning an operation for *gastric* ulcer, the gastric secretion tests give some guidance. True achlorhydria, when associated with a gastric ulcer, is strong evidence that the ulcer is malignant; but if acid is secreted at all it does not help to distinguish between a malignant gastric ulcer and a benign gastric ulcer.

If the pentagastrin test provokes secretion in the range of that consistent with duodenal ulcer, essentially a duodenal ulcer diathesis (Johnson's type II or type III) may be strongly suspected.[30] This test does help the surgeon decide that the operation should be one of the duodenal ulcer type; that is, a vagotomy is probably indicated, whereas a partial gastrectomy with Billroth I reconstruction with vagotomy would be inappropriate.[31] A vagotomy with drainage, or a vagotomy and antrectomy would be more likely to give permanent relief.

With respect to *duodenal* ulcer, many investigators have stated that the pentagastrin test is not likely to be helpful in the selection of an operation. Nevertheless, Kronborg[5] is the exception in finding the tests of value in choosing the appropriate operation. He recommended that men with a PAO of 46.4 mg/h or greater and women with a PAO of 42.2 mg/h or greater should have a vagotomy and antrectomy, because the rate of recurrence in these patients following vagotomy and drainage was significantly greater than in those with lesser gastric secretion values. His study was a careful prospective trial and the results have greater validity than the opinion based on some other less well-controlled studies. Moreover, Bock, du Toit and Boyd,[32] in determining prognosis, have found that men less than 30 years of age with a high MAO are more likely to require operation than those in whom the MAO lies in the normal range.

Watery Diarrhea, Hypokalemia, Achlorhydria Syndrome (Verner-Morrison Syndrome)

This syndrome, like the Zollinger-Ellison syndrome, is associated with a pancreatic tumour that is not of the B-cell type. Profuse watery diarrhea, hypokalemia and achlorhydria or hypochlorhydria are the clinical features. Basal achlorhydria is present in 60% of patients and histamine stimulation fails to produce a response in 30%. Hence, gastric acidity studies in patients with profuse diarrhea and hypokalemia will assist in making a diagnosis.[33]

Conclusions

Tests of gastric secretion may be found useful in the following circumstances:

1. In patients with ulcer-like dyspepsia, when endoscopic and radiologic findings are normal or equivocal, stimulated secretion may be high enough to be diagnostic of duodenal ulceration.

2. In patients with gastric ulceration, stimulated output may be high enough to indicate that the patient has a duodenal ulcer diathesis. If there is true achlorhydria a gastric ulcer is malignant, but otherwise the test does not distinguish a malignant ulcer from a benign ulcer.

3. The tests of stimulated secretion may be of value in selecting the operation for peptic ulceration, in choosing the operation for peptic ulceration or in predicting the eventual need for operation.

4. In patients who have recurrent symptoms after operation for peptic ulceration and in whom the radiologic and endoscopic examinations are inconclusive, the tests may be of help in deciding whether there is a recurrent ulcer. They also may give some

direction in planning operation for recurrent ulceration.

5. The tests are of help in suspecting the presence of Zollinger-Ellison syndrome.

6. The tests may be helpful in diagnosing retained antrum in patients who have had a gastric resection and have recurrent ulceration.

7. The tests will be helpful in diagnosing the Verner-Morrison syndrome.

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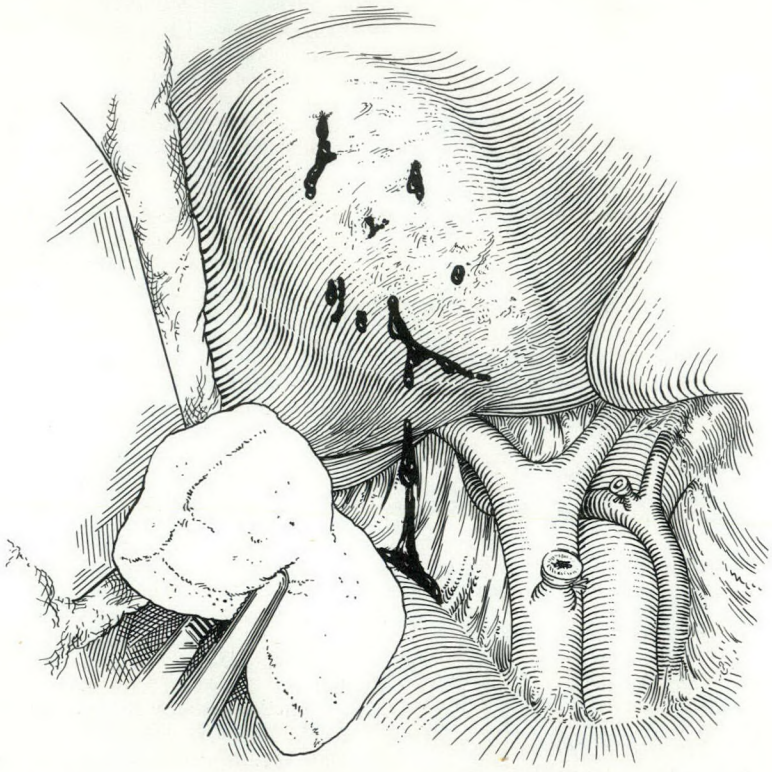

still the only absorbable hemostat that meets all these criteria:

- absorbed completely in four to six weeks
- safe as a permanent packing in any tissue; won't interfere with bone regeneration, cause cyst formation or excessive scar tissue
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- stimulates natural clotting and healing of disrupted tissue

POST-TRAUMATIC PULMONARY INSUFFICIENCY FOLLOWING CHEST TRAUMA: IDENTIFICATION AND PROPHYLAXIS*

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Summary: Post-traumatic pulmonary insufficiency is a syndrome characterized by tachypnea, increasing respiratory effort, progressive pulmonary infiltrates and relentless progressive hypoxemia. Prophylaxis of this syndrome currently comprises adequate treatment and depends upon its early recognition by frequent monitoring of blood gases, chest radiograph, and determination of values of circulatory variables. Once the syndrome is recognized in its early stages, artificial ventilation, with the possible addition of positive end-expiratory pressure, meticulous fluid therapy properly monitored, and prevention of infection constitute the hallmark of treatment.

Résumé: L'insuffisance pulmonaire post-traumatique est un syndrome qui est caractérisé par de la tachypnée, un effort pulmonaire croissant, des opacités pulmonaires progressives et une hypoxémie progressive sans rémission. Le traitement prophylactique de ce syndrome comporte un traitement adéquat et dépend essentiellement de son diagnostic précoce au moyen de fréquentes analyses des gaz du sang, de radiographies thoraciques et de l'analyse des paramètres circulatoires. Une fois ce syndrome identifié dans sa phase initiale, le traitement comporte essentiellement la respiration artificielle, avec, le cas échéant, le recours à la pression positive en fin d'expiration, l'administration méticuleuse et convenablement surveillée de sérums artificiels et la prévention de l'infection.

THE immediate treatment of chest trauma is now fairly standard in all major centres. A substantial proportion of the morbidity and mortality attributed to chest injury is due to late complications such as renal failure, thromboembolism, sepsis and post-traumatic insufficiency.[1] This paper is concerned with post-traumatic pulmonary insufficiency. It is an interesting entity, as evidenced by the frequent review articles appearing in the recent literature.[1-7]

Post-traumatic pulmonary insufficiency, also known as adult respiratory distress syndrome, shock lung, and wet lung, among other synonyms,[4] is characterized by tachypnea, increasing respiratory effort and, on auscultation, lungs that usually sound dry initially but become "wet" as the syndrome progresses. Within 24 to 48 hours chest radiographs show progressive infiltrates, which then may develop into major consolidation. Radiographically, the fully developed syndrome is characterized by a "white-out" appearance with very little evidence of aerated lung. Accompanying this is a relentless, progressive hypoxia that worsens even as the inspired oxygen concentration is increased.

Implicit in this description is the understanding that other causes of progressive hypoxia have been sought and excluded. These include left heart failure, aspiration pneumonia, bronchopneumonia, atelectasis, pulmonary embolism, pulmonary contusion and pneumothorax. The early identification of this perplexing entity is the main hope of preventing an eventual fatal outcome; this depends on close, frequent monitoring and a high degree of suspicion.

IDENTIFICATION

Blood-gas analysis is essential, and every patient who has sustained severe chest trauma should have an indwelling arterial catheter. This will aid in the frequent monitoring of blood gases, particularly Pao_2. Initially the Pao_2 is stable in the presence of a constant FIo_2. Subsequently, a decreasing Pao_2, even though the FIo_2 is constant or increasing, should immediately bring to mind the possibility of post-traumatic pulmonary insufficiency.

Chest radiographs should be taken frequently during the first 48 hours after injury. The development of new, reticular infiltrates during this period is ominous.

The discovery of pulmonary infiltration

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is usually preceded by a decrease in Pao_2, because the former constitutes evidence of the gross pericapillary hemorrhage and exudate that is the hallmark of this disease, whereas the decreasing Pao_2 is evidence of more subtle early changes.

Clinically, a gradually rising respiratory rate is associated with a corresponding increase in the effort expended by the patient. An increasing use of accessory muscles of respiration, grunting, sweating, cyanosis and tachycardia will generally be noted. In addition to using these signs in monitoring the respiratory status, one must also continuously monitor the circulatory status. Blood pressure, pulse rate, urinary output and either right atrial or pulmonary capillary wedge pressure as a reflection of left atrial pressure should also be examined. The ease of insertion of the Swan-Ganz catheter has led to its increased use in monitoring wedge pressure, and it has enabled us to rule out left heart failure, fluid overload in the initial resuscitative effort, as well as pulmonary embolism as a cause of the falling Pao_2.

Moore and his colleagues[8] have outlined four stages in the development of post-traumatic pulmonary insufficiency. However, these phases developing after chest injury are rarely distinct. Phase 1 with alkalemia and absence of hypoxia rarely occurs, if ever. Phases 2, 3 and 4—circulatory stability and hypoxemia, then acidosis and progressive hypoxia, and finally, terminal hypoxia and hypercarbia—are not distinct and, in fact, in many cases do not occur.[9]

Prophylaxis

The cornerstone of treatment is prophylaxis, which depends on an understanding of the pathophysiologic aspects of the disease. Although there is still much controversy about the etiology of this syndrome,[10] the possible causes can be divided arbitrarily into two groups. The effects of the first group of causative factors are not readily prevented by medical means. In various combinations, platelet microemboli, disseminated intravascular coagulopathy, vasoactive peptides, serotonin, histamine, kinins, prostaglandins and fat emboli have been implicated.[4] These factors, possibly released by injured tissue or by hypoperfusion of the lung during a period of hypotension, increase capillary permeability and allow fluid to leak into the interstitium of the lung; the result is the heavy congested lung that is seen at autopsy. This defect in the architecture of the lung leads to a deterioration in gas exchange.

Although it is difficult to conceive of any method of forestalling the first group of events, the consequences of a second group of possible causative factors can be prevented. This group includes microembolization of blood-transfusion particles into the pulmonary circulation, oxygen toxicity, inappropriate fluid therapy in the resuscitation period (either too much or too little) and sepsis. For these factors the following regimen is recommended:

"Don't vacillate, ventilate!".—When the evidence points to post-traumatic pulmonary insufficiency, an endotracheal tube should be passed and artificial ventilation begun. It is at this early stage that prevention is possible. If severe hypoxia and acidosis are allowed to develop, the resulting pulmonary vasoconstriction will compound the already worsening state. Once the initial period of resuscitation is over, the time has come to "walk the oxygen tightrope".[11] As soon as the blood-gas results are available the Fio_2 should be adjusted to give a Pao_2 between 80 and 100 mm Hg. Should this require an oxygen concentration greater than 40%, the inspired oxygen concentration should be adjusted to give a Pao_2 of between 60 and 80 mm Hg. The lower Pao_2 is acceptable in view of the advantage of not exposing the alveoli to an inspired oxygen concentration of greater than 60% and running the risk of oxygen toxicity. If a Pao_2 exceeding 70 mm Hg cannot be maintained with less than 60% inspired oxygen, positive end-expiratory pressure (PEEP) should be used.[12] One should begin with PEEP of 5 cm H_2O and increase to a pressure of 10 cm H_2O or even 15 cm H_2O if necessary to keep the Pao_2 above 70 mm Hg while administering an oxygen concentration of less than 60%. PEEP will keep patent alveoli that would otherwise be closed during expiration; these alveoli are thus kept open during the entire respiratory cycle. In effect, the functional

residual capacity is increased and the venous admixture is decreased, which causes the Pao_2 to increase at a constant inspired oxygen concentration. This has two major advantages in the prophylaxis of post-traumatic pulmonary insufficiency: first, the inspired oxygen concentration can be maintained below 60%, which limits the possibility of oxygen toxicity; second, the increase in the mean intra-alveolar pressure leads to a decrease in the hydrostatic pressure gradient from the pulmonary capillary to the alveolus, and thus counteracts the transudation of fluid from the pulmonary circulation into the alveoli. The use of PEEP, however, is not without hazard: pneumothorax is always possible and in the hypovolemic patient, cardiac output may decrease.

Monitor the circulatory status and restore perfusion.—In every patient with a serious chest injury one should insert either a central venous pressure catheter, for monitoring of right atrial pressure,[13] or a Swan-Ganz catheter for monitoring of pulmonary wedge pressure. In the initial resuscitative period crystalloid fluid should be infused until type-specific blood is available. Once this is available it should be infused as needed. The blood should be as fresh as possible and infused through a Micropore filter in order to decrease microembolization of particles into the lung. The end point of resuscitation should be the restoration of an adequate urinary output (greater than 30 ml/h), restoration of normal right or left venous pressure and good peripheral perfusion. Attempts to restore the blood pressure to normal pre-injury values are not indicated as long as peripheral perfusion is adequate. Although overenthusiastic resuscitative efforts have been implicated as being one of the possible causes of post-traumatic pulmonary insufficiency due to fluid overload with pulmonary edema, there is evidence that keeping the patient too "dry" is also not optimum.[4] If pulmonary edema does develop despite normal or low left atrial pressures the infusion of albumin and the administration of furosemide (Lasix) has been advocated.[14]

Prevent infection.—Debridement of all wounds must be adequate; no traumatized or necrotic tissue must remain. The simple rules of good respiratory care must be followed. Frequent changes of nebulizers, humidifiers and respiratory tubing, and application of an aseptic suction technique and prevention of crossinfection are all important. Gram's staining and sputum cultures of the endotracheal or tracheostomy tube aspirate should be done daily in order to identify colonization. Antibiotics, however, should not be used unless there are clinical signs of infection;[15] prophylactic antibiotic therapy should be avoided. As well as meticulous respiratory care all indwelling catheters must receive special attention and be inserted under strict aseptic technique.

Administer steroids.—Massive doses of steroids (30 mg/kg of methylprednisolone hemisuccinate) have been advocated to prevent subcellular disorganization within the lung.[16] Steroid therapy seems most effective if the drug is given soon after the occurrence of the shock-producing trauma. The use of steroids, however, is still controversial.[3, 4]

Prophylaxis is an important part of the treatment of post-traumatic pulmonary insufficiency because if the measures that have been outlined fail to prevent or arrest the relentless progression of hypoxia the only remaining form of treatment is temporary cardiopulmonary bypass with a membrane oxygenator. And this form of treatment, which is obviously a temporary measure of benefit to the patient while the pulmonary condition improves, has proved disappointing in the isolated instances in which it has been used.[9]

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RADIOLOGIC FEATURES OF FAT EMBOLISM SYNDROME

Diagnosis of fat embolism syndrome may be made on the basis of: (a) pulmonary and cerebral dysfunction in a patient with, most frequently, a history of a long-bone fracture in whom the chief manifestations are fever and abnormal arterial blood-gas values and (b) one or more of the following: petechiae, fat in the retinal vessels, fat in the urine, fat on cryostat frozen section of clotted blood, and an abnormal chest radiograph (Dines, Linscheid and Didier, Mayo Clin Proc *47: 237, 1972). Among the features of this syndrome described by Feldman, Ellis and Green* (Radiology *114: 535, 1975) are the radiographic findings.*

These findings are as follows: For the first 72 hours or longer, the lungs may appear grossly normal. Generalized lung density then develops, though the apices are spared. The density, at first more apparent in the perihilar and basilar areas, comprises a combination of interstitial and alveolar patterns. These findings may be regarded as being characteristic, though they are nonspecific. The density usually clears after several days.

When assisted ventilation is required, the central and the larger air ducts, and also more fully ventilated or emphysematous peripheral air spaces, become evident and stand out against the surrounding opaque lung. These patchy, lucent areas may be particularly noticeable during positive-pressure ventilation owing to the pressure effects and heterogeneous distribution of forced ventilation.

Feldman, Ellis and Green also review the incidence and pathophysiology, laboratory abnormalities, clinical features and treatment of the fat embolism. Their paper also gives details of the etiology and clinical features of nine of their own cases of post-tramautic fat embolism.

EFFECT OF FUNDOPLICATION ON THE LOWER ESOPHAGEAL SPHINCTER*

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Summary: The lower esophageal sphincter of 26 patients has been studied by infusion manometry before and after a modified Nissen fundoplication. Of these patients, 24 have remained asymptomatic (mean follow-up, 21 months). The mean time for a positive acid infusion test increased from 267 seconds before, to 617 seconds after operation. The common cavity test was positive in 22 of the 26 patients before operation but positive in only the 2 symptomatic patients after operation. Mean lower esophageal sphincter pressure (cm H_2O) increased from 12.6 ± 2.0 to 16.5 ± 1.5 ($\bar{x}$ ± SEM). However, in only 15 of the 26 patients was an increase in lower esophageal sphincter pressure demonstrated and in 7 a decrease in pressure was noted. One patient's symptoms recurred at 3 months in spite of an increase in lower esophageal sphincter resting pressure of 8 cm H_2O. The other symptomatic patient demonstrated a rise in lower esophageal sphincter pressure at 3 months of 8 cm H_2O, though this decreased to preoperative levels at 26 months, the time of symptom recurrence.

Intraluminal pressure of the human cadaver esophagus after removal from the body was studied by means of an infusion manometric technique. Narrowing the esophageal lumen with extrinsic weight of as little as 50 g resulted in a zone of elevated pressure. The amplitude of the elevated pressure zone was directly related to the infusion rate.

Elevations in lower esophageal pressure following fundoplication are probably secondary to extrinsic compression and are not necessary for a good clinical result.

Résumé: Avant et après avoir pratiqué, chez 26 malades, l'opération modifiée de Nissen (plicature du fond gastrique sur le sphincter inférieur de l'œsophage) nous avions étudié le sphincter œsophagien inférieur par une infusion assortie d'une mesure manométrique. De ces 26 malades, 24 sont demeurés asymptomatiques (en moyenne pendant 21 mois). Le délai moyen pour la positivité de l'épreuve d'infusion acide est passé de 267 secondes avant l'opération à 617 secondes après l'intervention. De même, l'épreuve de la cavité commune était positive chez 22 des 26 malades avant l'opération, mais n'était positive que chez 2 des malades symptomatiques après l'opération. La pression moyenne régnant dans le sphincter œsophagien inférieur (exprimée en cm de H_2O) était passée de 12.6 ± 2.0 à 16.5 ± 1.5 ($\bar{x}$ ± ESM). Néanmoins, seulement chez 15 des 26 malades, on notait un accroissement de la pression intrasphinctérienne et une diminution de la dite pression chez 7 autres. Chez un malade, les symptômes ont réapparu 3 mois après l'opération, malgré une augmentation de la pression intrasphinctérienne au repos de 8 cm H_2O. Chez l'autre malade symptomatique on notait une augmentation de la pression intrasphinctérienne de 8 cm H_2O à 3 mois, celle-ci étant cependant redescendue aux pressions préopératoires notées 26 mois auparavant, ce qui coincidait avec la résurgence des symptômes.

Nous avons mesuré la pression existant dans la lumière d'un œsophage de cadavre, après son ablation et avons utilisé à cette fin, la technique de l'infusion manométrique. Le rétrécissement de la lumière de l'œsophage par un poids extrinsèque de seulement 50 g, s'est traduit par une zone d'augmentation de la pression. L'amplitude d'augmentation de la zone de pression était directement proportionnelle à la vitesse de l'infusion.

Les augmentations de la pression intrasphinctérienne de l'œsophage après l'opération de Nissen modifiée sont vraisemblablement attribuables à une compression extrinsèque et elles ne sont pas obligatoires pour réaliser un bon résultat clinique.

SEVERAL reports attest to the efficacy of fundoplication in relieving the symptoms of gastroesophageal reflux.[1-4] The most reasonable explanation for the success of fundoplication is that a simple mechanical flap valve is formed. Production of gastroesophageal reflux in the cadaver is virtually impossible following fundoplication.[5]

There are, however, reports that suggest

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a more physiologic effect of fundoplication. Increases in the resting tone of the lower esophageal sphincter (LES) have been observed.[6-8] In addition, after fundoplication there is an improvement in the response of the manometric LES to abdominal compression and pentagastrin.[8]

In this investigation we examined the LES manometrically before and after fundoplication. We attempted to determine whether the alleviation of symptoms after fundoplication can be attributed to either creation or improved function of a physiologic sphincter.

Materials and Methods

Patients

Fundoplication was performed in 26 patients for severe, recurrent heartburn and regurgitation persisting in spite of adequate medical management. A hiatus hernia was demonstrated in 20 of the 26 patients by barium meal, and endoscopic changes suggestive of esophagitis were observed in 14.

Operative Procedure

In all patients the surgical approach was intra-abdominal and an 80%- to 90%-circumferential, modified Nissen fundoplication, similar to that described by Lind, Burns and MacDougall,[7] was performed. After the gastroesophageal junction had been mobilized, the posterior aspect of the gastric fundus was passed behind the esophagus and sutured to the right anterior margin of the esophagus over a 4- to 6-cm distance. The anterior fundus was then sutured to the left anterior margin of the esophagus so as to leave a 1-cm defect between the suture lines (Fig. 1). The patients were followed-up by personal interview at 3 and 6 months and thereafter at 6-monthly intervals for 1 to 3 years (mean follow-up, 21 months).

Manometric Studies

Each patient underwent a motility study before the operation and 3 months after the operation. In patients who became symptomatic after 3 months another motility study was repeated at that time.

Motility studies were performed with a catheter assembly consisting of four fused polyethylene catheters (ID, 0.047 in; OD, 0.067 in). Each catheter had a lateral orifice equal in diameter to the tube itself and was plugged immediately distal to this opening. The holes were located 5, 1 and 5 cm apart and were oriented circumferentially so that the orad three openings lay at 120° to each other. Each catheter was connected to an external Statham P23De pressure transducer and continuously perfused with water during the resting study at 1.8 ml/min and during the deglutition studies at 3.6 ml/min. All subjects were studied after a 12-hour fast. The tube assembly was inserted into the stomach and, after a rest of 15 minutes, the test began.

Resting sphincter pressure.—The tube assembly was withdrawn 0.5 cm at a time so that the orad three openings traversed the distal zone of elevated pressure and entered the esophagus. The patient was instructed not to swallow during this portion of the study and continuous electromyographic monitoring of pharyngeal musculature allowed continuous study of this variable. The tube assembly was left at each station until the pressure stabilized; this always took a minimum of 15 seconds, but occasionally took up to 90 seconds.

Response of the LES to abdominal com-

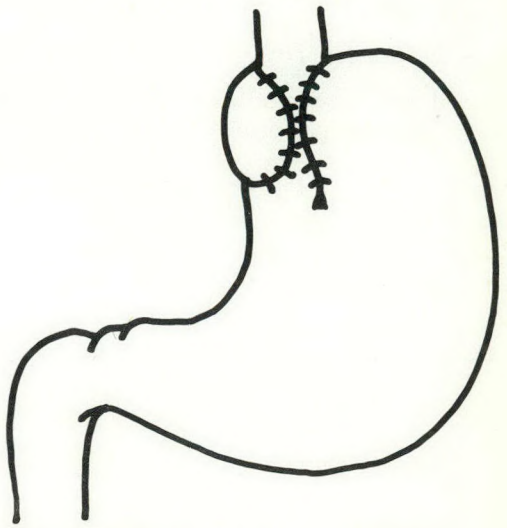

Fig. 1.—Modified Nissen fundoplication. Gap of unwrapped esophagus (1 cm) was left between two suture lines.

pression.—The assembly was reinserted and abdominal compression (50 mm Hg) was applied by means of an enlarged thigh blood-pressure cuff placed about the upper abdomen and held in position with a Scultetus abdominal binder. The pull-through of the first three catheters was then repeated under identical conditions. The binder was removed and the tubes reinserted into the stomach.

Deglutition study.—A third pull-through at 0.5-cm intervals was then performed. At each interval the response of the LES to a 5-ml bolus of water swallow was then determined (Fig. 2).

Common cavity test.—Simultaneous increases in gastric and esophageal body during abdominal compression have been described in patients with gastroesophageal reflux.[7] A four-catheter manometric catheter assembly, with the opening of each tube 5 cm apart, was positioned to allow simultaneous recording of pressures from stomach, lower esophageal sphincter and body of the esophagus at both 5 and 10 cm above the LES. Abdominal compression (50 mm Hg) was then applied and the response of the esophagus, stomach and LES observed. The pressure was then released and, after a 1-minute rest, greater abdominal compression (80 mm Hg) was applied. A positive test was indicated if an elevation in distal esophageal pressure was observed in either of the tests.

Acid Infusion Test

Following the motility test an acid infusion test was performed. The manometric catheter assembly was positioned as during the common cavity test. Saline was infused for 5 minutes through the proximal tube (10 cm above the lower esophageal sphincter) at 8 ml/min. The infusion solution was then changed to 0.1N hydrochloric acid without the patient's knowledge and the time to reproduce exactly the patient's symptoms was determined. The solution was then changed again to saline and the infusion was continued until the patient's symptoms were relieved. The test was considered positive if the patient's primary symptoms were reproduced.

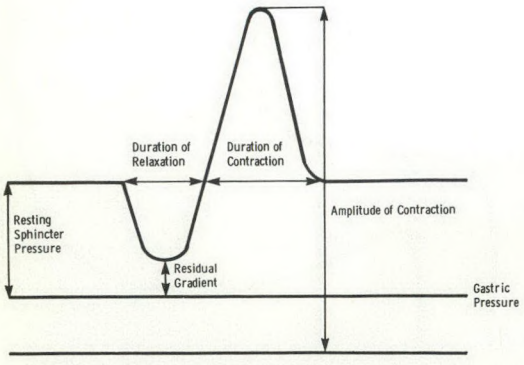

Fig. 2.—Diagrammatic representation of response of lower esophageal sphincter to deglutition.

Cadaver Esophagus Study

Human cadaver esophagus was studied after removal from the body. In different studies, weights of 50, 75, 100 and 125 g were applied to a 3-cm area of the esophagus by means of a beaker filled with various amounts of water. A manometric catheter assembly was then pulled through the esophagus at 1 mm/sec. Water was infused through the catheters during the different studies at 0, 0.7, 1.8, 3.6, 7.2 and 18 ml/min.

Results

Complete resolution of symptoms noted preoperatively occurred in 24 of the 26 patients. Symptoms recurred in one patient 3 months after operation and in another, after 26 months.

The common cavity test was positive in 22 of the 26 patients before operation. After fundoplication the test was positive only in the two patients in whom the symptoms recurred.

The acid infusion test was positive in 25 of 26 patients before operation (mean time, 267 $\pm$ 77 [$\bar{x} \pm$ SEM] seconds). After fundoplication there was in all 26 patients an increase in the time required for reproduction of symptoms, and in 7 the test became negative; the mean time for a positive test following operation was 616 $\pm$ 89 ($\bar{x} \pm$ SEM) seconds ($P < 0.01$).

The mean resting sphincter pressure increased from 12.6 $\pm$ 2.0 H_2O before operation to 16.5 $\pm$ 1.5 cm H_2O after oper-

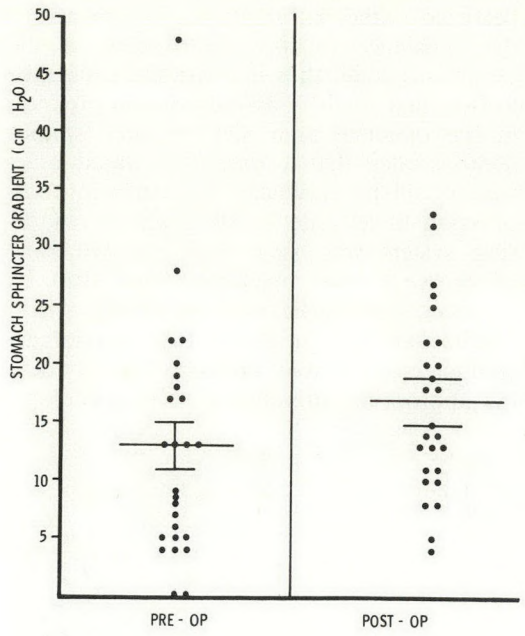

Fig. 3.—Lower esophageal sphincter pressure (stomach sphincter gradient) before and after Nissen fundoplication (mean ± SEM).

ation ($P < 0.01$) ($\bar{x} \pm$ SEM). However, in only 15 of the 26 patients was there an increase in LES pressure, in 4 there was no change and in 7 a fall in pressure was noted (Fig. 3).

With respect to the patient whose symptoms recurred at 3 months, sphincter pressure increased from 0 cm H_2O before operation to 8 cm H_2O at the time of recurrent symptoms. In the patient with recurrent symptoms 26 months after operation, sphincter pressure increased at 3 months from 18 cm H_2O before operation to 26 cm H_2O, and by 26 months sphincter pressure had returned to the preoperative level of 18 cm H_2O.

No improvement was observed in the response of the LES to abdominal compression after operation (Fig. 4). The ratio of change in sphincter pressure to change in gastric pressure was 0.8 before operation and 1.0 after fundoplication ($P > 0.05$).

Gastric pressure increased from 12.0 ± 1.3 cm H_2O before to 16.6 ± 1.0 cm H_2O after operation ($\bar{x} \pm$ SEM).

Several changes were observed in the re-

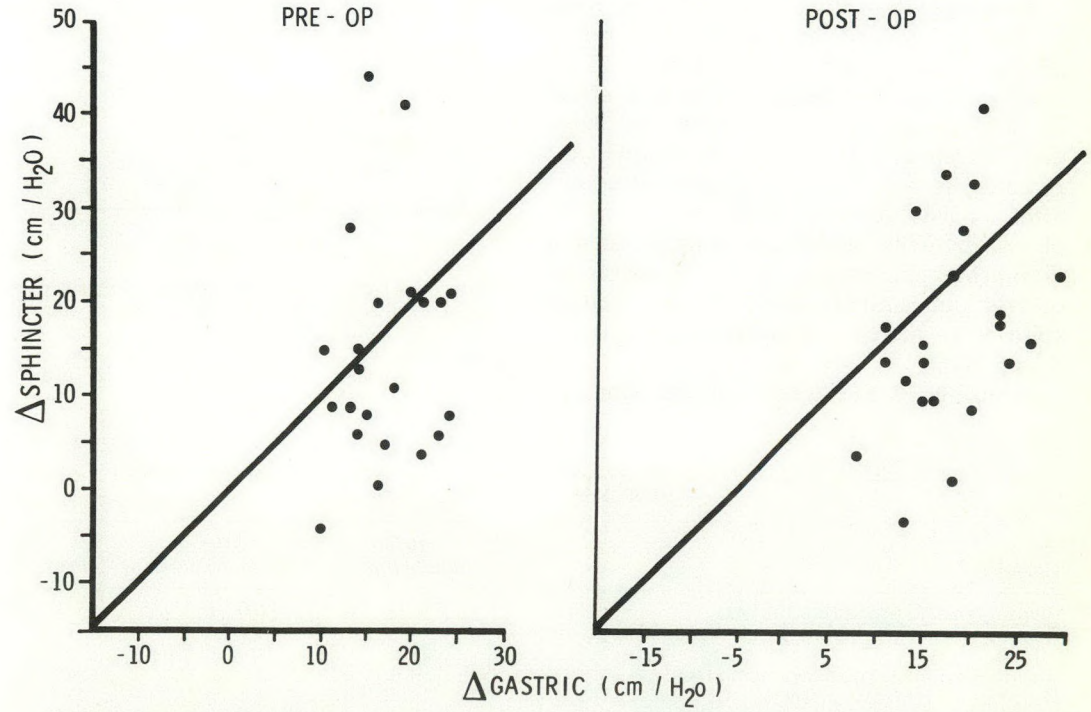

Fig. 4.—Response of LES to abdominal compression before and after operation. Ratio △ sphincter pressure:△ gastric pressure increased only from 0.8 to 1.0 ($P > 0.5$).

sponse of the LES to deglutition (Table I). The residual stomach sphincter gradient during relaxation increased significantly from 1.4 ± 0.5 cm H_2O to 4.4 ± 0.8 cm H_2O. The amplitude of sphincter contraction increased, also significantly, from 12.2 ± 1.3 to 10.7 ± 1.0 seconds.

Extrinsic compression of a cadaver esophagus produced a zone of elevated pressure. This zone of elevated pressure was observed only during perfusion of catheters. The magnitude of the pressure was related directly to the rate of perfusion and to the weight applied to the esophagus (Fig. 5).

Discussion

The fundus of the stomach is similar to the LES in its ability to relax actively during deglutition.[9] It might then seem reasonable that supporting the distal esophagus by the gastric fundus, as is done in fundoplication, would create a physiologic sphincter. Support for such a concept is provided by studies that demonstrate an elevation in LES resting pressure and improved responsiveness to pentagastrin and abdominal compression after a surgical procedure.[8]

However, Butterfield[5] was able to prevent artificially induced gastroesophageal reflux in the cadaver after fundoplication. This strongly suggests a purely mechanical effect of the operation. In this study we have demonstrated that an increase in sphincter pressure is not necessary to prevent symptoms, at least over the short term. In seven of our patients we in fact demonstrated a diminished sphincter pressure. Moreover, in one of our patients symptoms recurred in spite of an increase in sphincter pressure of 8 cm H_2O.

Suturing of the fundus of the stomach about the esophagus probably results in some degree of esophageal narrowing. A perfused catheter system largely measures the resistance to flow. Narrowing of the esophagus could thus increase the resistance to flow and result in an elevation in pressure in the operated area. Our *in vitro* studies demonstrated that a zone of elevated pressure could be produced by extrinsic compression of as little as 50 g when an infusion system was used. Any elevated zone of pressure after operation must then be reviewed with suspicion.

The universal property of all physiologic sphincters is the ability to relax actively during appropriate stimulation. If a physiologic

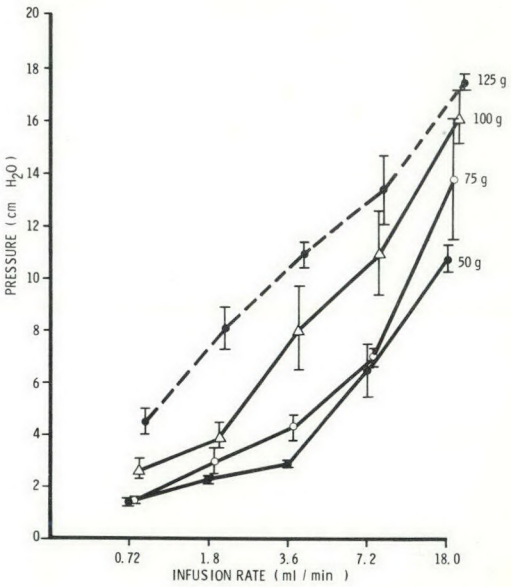

Fig. 5.—Effect ($\bar{x}$ ± SEM) on intraluminal pressure of localized extrinsic compression on cadaver esophagus. Zone of elevated pressure was produced with extrinsic weight as small as 50 g. Pressure recorded was directly related to infusion rate.

TABLE I.—Effect of Fundoplication on the Lower Esophageal Sphincter Response to Deglutition

Variable	*Before fundoplication*	*After fundoplication*	*P value*
Resting gastric pressure (cm H_2O)	12.0 ± 1.3	16.6 ± 1.0	<0.05
Resting stomach — sphincter gradient (LES pressure [(cm H_2O)]	12.6 ± 2.0	16.5 ± 1.5	<0.01
Residual gradient relaxation (cm H_2O)	1.4 ± 0.5	4.4 ± 0.8	<0.5
Duration of relaxation (sec)	9.9 ± 0.8	11.5 ± 0.9	0.3
Amplitude of contraction (cm H_2O)	37.8 ± 3.7	55.2 ± 5.7	<0.01
Duration of contraction (sec)	12.2 ± 1.3	10.7 ± 1.0	0.03

sphincter is created, relaxation should compensate for any increased sphincter pressure. We observed a mean increase in resting pressure at 4 cm H_2O. The residual gradient between sphincter and stomach increased by 3 cm H_2O.

We suggest that increases in sphincter pressure after fundoplication are probably secondary to extrinsic narrowing and do not constitute evidence that a physiologic sphincter has been created. Moreover, such increases in sphincter pressure are probably not necessary for the creation of a competent gastroesophageal junction.

The authors acknowledge the assistance of Mrs. Diane Brown, RN, in these studies.

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GLIMPSES OF SURGICAL HISTORY: D FOR DIATHERMY

D. A. E. SHEPHARD

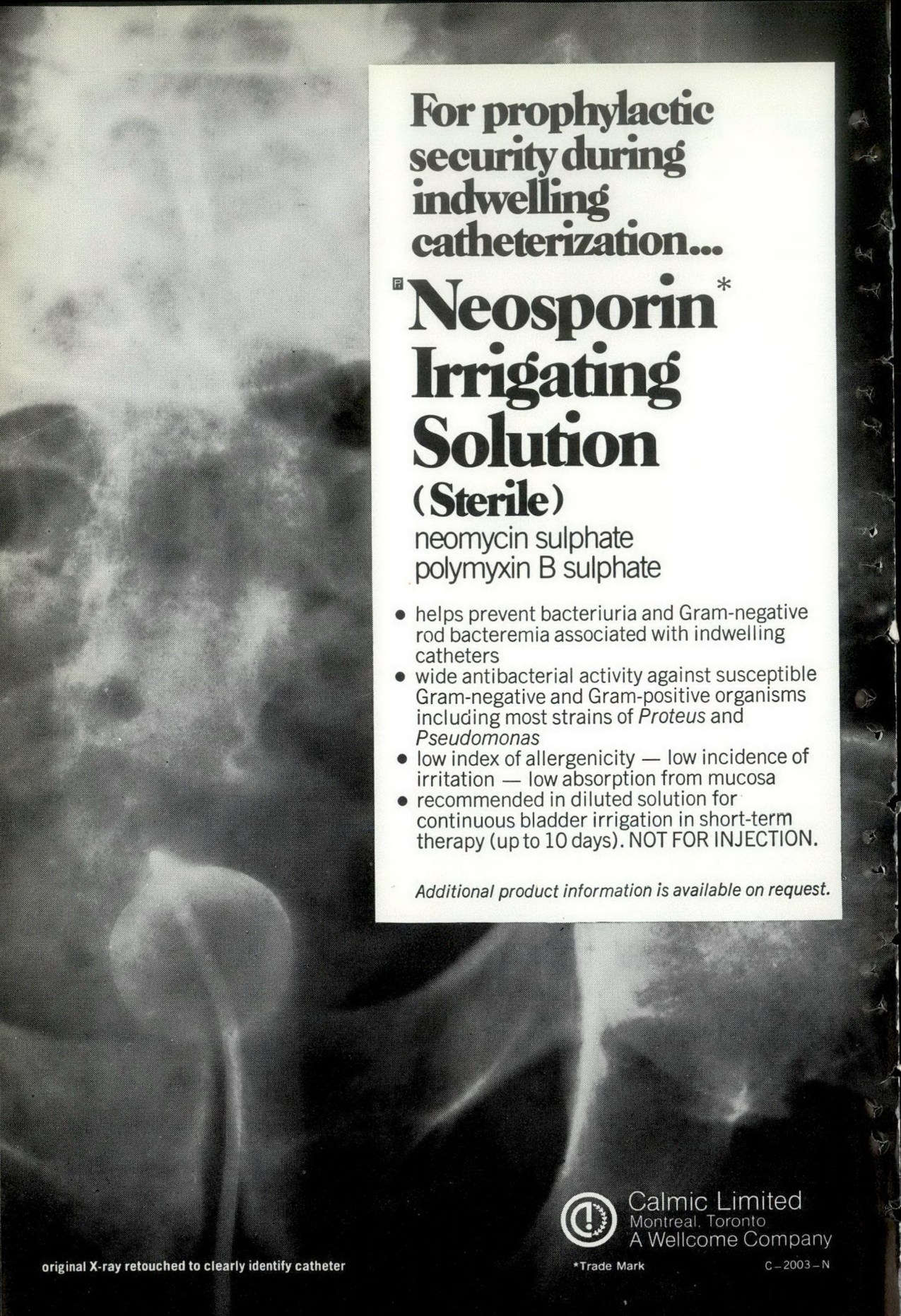
Heat, like light, has always been therapeutically beneficial. Blankets, sweat baths, hot water bottles and heated stones have all been used—and even "the lively vigorous warmth of young people," like the virgin who lay in old King David's bosom. In surgery, heated instruments were used despite the pain and the crudity of the method, but it was not until comparatively recent times that surgeons acquired a useful scientific method of heat production: DIATHERMY.

Harvey Cushing was one of many surgeons early in the 20th century who found that diathermy coagulation improved surgical results. He wrote in 1927 of the diathermy electrode that "it constitutes a surgical tool which bids fair to replace the scalpel . . .". But for the historian of surgery, diathermy has another interest: it illustrates the theme of this A-to-Z series—that the practice of surgery has frequently advanced as a result of work in totally unrelated disciplines.

In 1891, the physicist Tesla observed that the passage of high-tension, high-frequency electric currents through the body produced heat. In the nonmedical journal Electrical Engineer, *he suggested the use of high-frequency currents for medical practice. And in 1910, in another electrical journal,* Electrical World, *it was reported that Doyen had used electrically produced heat in the treatment of cancer. (Cancer cells were destroyed by heat and the need for the surgical knife, it was reported, might be eliminated.)*

This period, 1891 to 1910, was an active one in the development of diathermy. In 1898 d'Arsonval, a professor of medical physics in Paris, had started to use electric currents in investigating the therapeutic effects of high-frequency currents, and many other workers followed him. Outside medicine a critical development was Marconi's work in wireless telegraphy; he transmitted signals across the Atlantic (from Canada to England) in 1901, and others followed him too. But wireless telegraphy required powerful high-frequency currents, and thus appropriate apparatus was developed. Soon medical and surgical apparatus revealed similarities to apparatus used in wireless telegraphy. The heat produced by these refined high-frequency currents was large; in fact, there was a "heating through" of the tissues—a fact led Nagelschmidt in 1908 to coin the term "diathermy".

Let us return to Cushing. In a 1928 paper, Cushing's coauthor was a physicist, Bovie. They reported on the use of the Bovie apparatus in neurosurgery; and the Bovie apparatus was a factor in Cushing's improved results of delicate neurosurgical operations. Cushing, the surgeon, collaborated with Bovie, the physicist—one of a number of useful symbiotic partnerships that have benefited the practice of surgery and surgical patients too.



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THORACICOABDOMINAL TRAUMA: A PLAN FOR INITIAL MANAGEMENT*

CLEMENT A. HIEBERT, MD†

Summary: Trauma to multiple organs damages the body but it also disrupts the orderly process of history taking, physical examination, laboratory work, calm deliberation and treatment. The need for a rehearsed resuscitation protocol is important, as is a systematic scheme that emphasizes the skilful use of eight specific cannulas and the treatment of priority problems first.

Résumé: Des traumas atteignant de multiples organes, non seulement lèsent l'organisme, mais aussi dérangent gravement les méthodes courantes qu'on emploie pour décider de la marche logique du traitement, comme l'histoire du cas, l'examen somatique, le travail du laboratoire, et les délibérations multidisciplinaires qui se font dans le calme. En pareil cas, il faut de toute évidence mettre au point un protocole de réanimation détaillé et un plan systématique qui insiste sur l'emploi pudicieux de huit canules spécifiques et sur l'ordre qui doit régir la priorité des problèmes essentiels.

DURING 1975 it is expected that surgeons in North America will have to care for more than 2 million victims of unplanned automobile deceleration.[1] Approximately 55 000 of these patients in the United States and 6000 in Canada[2] will sustain such severe organ damage that either they will be dead on arrival at hospital or will die while their injuries are being diagnosed and treated.

Multiple trauma disrupts not only organs but also organizations. Among the latter are the emergency rooms of many hospitals. There are three reasons for disruption of organization: (a) the gravity of the patient's condition demands commensurately rapid and simultaneous history, examination and treatment, in contrast to the leisurely sequential process appropriate for a patient with a chronic illness; (b) the response of physicians to the acute disability resulting from damage to several organ systems is akin to that in the management of a patient who has, concurrently, a collapsed lung, ruptured appendix and a bleeding ulcer; and (c) there is an unfortunate tendency to deal with obvious lesions first, particularly deformed bones and cut skin, at the intolerable expense of early identification of concealed vascular or visceral injuries.

To overcome these inherent pitfalls, the surgeon must not only become sensitized to each organ's distress signal but also prepare himself and his associates with an unfailing sense of priority and certain technical skills before the trauma patient arrives in the emergency room.

The scheme for managing multiple trauma that is presented in this paper presumes that at least two professionals are in attendance—one collecting and recording data, writing orders, and completing requisitions for radiographs and other studies, and the second engaging himself in actual treatment. The rationale of this system derives from the observation that the essential resuscitative effort depends on the swift and skilful introduction of as many as eight tubes into the airway, vessels and visceral spaces of the patient who has been seriously injured in an accident. These eight tubes are the following: an airway suction catheter; a bronchoscope; an endotracheal tube; a thoracotomy drain; a plastic intravenous catheter; a nasogastric tube; a bladder catheter; and a peritoneal lavage catheter (Fig. 1). Use of these tubes, or considera-

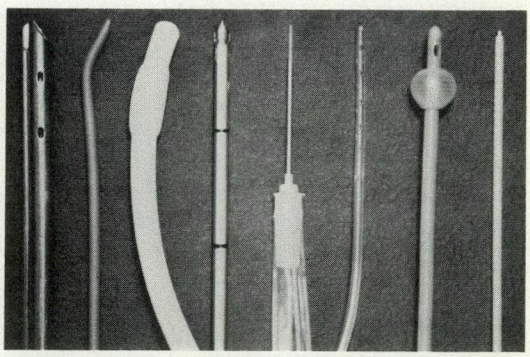

Fig. 1.—The eight essential tubes for initial resuscitation and treatment of the patient who has sustained multiple trauma. From left to right: bronchoscope, Coudé-tip catheter, cuffed endotracheal tube, thoracotomy tube, catheter for intravenous fluids and central venous pressure measurement, nasogastric tube, Foley catheter and peritoneal dialysis catheter.

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tion of their use, in a logical sequence means that most diagnostic, resuscitative and even therapeutic procedures will have been instituted or suggested. The ABC's of the suggested sequence follow the familiar pattern of the protocol for cardiopulmonary resuscitation.

The History

Ideally the history is best recorded by the member of the resuscitation team who is not supervising the actual treatment. In addition to ascertaining what happened and what parts of the body hurt, it is important to determine whether an unconscious patient was originally alert. The minimal essential history should also include the answers to the following questions: Is the patient a bleeder?, Does he have known allergy or idiosyncrasy to proposed medication?, Is he taking coumarin, insulin or cardiac drugs?, Has he been immunized against tetanus?, Does he have any known serious past illness?, When did he last eat?.

To record physical findings, outlines of the front and the back of the human figure provide an ideal place on which one can note not only lacerations and obvious traumatic lesions but also, for example, small contusions and bruises, which too frequently in retrospect are the only clues to important and otherwise concealed organ damage.

The Airway

A *Coudé-tip red rubber or plastic catheter* (size, no. 20 or larger) will permit one to remove secretions from the trachea by suction. It is nearly always possible to intubate the larynx by pulling the tongue forward and directing the tip of the catheter downward. Once secretions have been suctioned, oxygen is administered by mask.

A bronchoscope should always be available in the emergency room. It serves two purposes: first, the tubular bronchoscope not only provides an immediate airway but also allows one to introduce a large-calibre suction catheter and to recover aspirated material—and if a patient has vomited or if stomach contents are present in the pharynx, it must be assumed that food has been inhaled. Second, only bronchoscopy allows one to substantiate the possibility of a fracture of the proximal airway, an injury that is suggested by the presence of blood in the tracheal aspirate or prominence of mediastinal or subcutaneous emphysema. This very important lesion can be identified in 86% of instances by inspection of the bronchi through a conventional open-ended instrument.[3] The examination should take only a few minutes and the requisite skill should be acquired by every surgeon dealing with multiple trauma.

Breathing

A *cuffed endotracheal tube* connected to a volume-cycled respirator is recommended in the following four circumstances: chest wall injury with, either because of pain or paradoxical movement, impairment of lung expansion; parenchymal lung injury, as suggested by clinical, radiographic or blood-gas studies; depression of the central nervous system by trauma, alcohol or drugs; and superimposition of even apparently trivial chest injury on a chemically damaged airway, as, for example, with chronic cigarette smoking or aspiration of gastric contents. In each of these situations, neither clinical signs nor radiographs are as helpful as serial determinations of arterial oxygen tension.

A *thoracotomy tube* (size, no. 28 or larger) serves as both an indicator device and a means of definitive treatment in approximately 80% of instances of intrathoracic injury.[4] Its insertion from skin preparation to final dressing can be accomplished in less than 2 minutes if one is practised and the equipment kit is organized. The number of instruments should be limited to an absolute minimum. The prime indication for insertion of a chest tube is fluid or air in the pleural space. The brisk flow of blood that occurs immediately a thoracotomy tube has been inserted always looks alarming, but in practice it is only infrequently a cause for alarm. However, the immediate appearance of a litre or more of blood in the drainage bottle followed by continued bleeding (in excess of 250 ml/h) is an indication for surgery. In such a case, and in the case of a continuing massive air leak, the thoracotomy tube provides invaluable information on which to base decisions.

Circulation

A *polyethylene cannula* in the femoral artery and another in the subclavian or femoral vein provide routes for treatment as well as indicators of the success of that treatment. There is no place for the timid introduction of needles designed for scalp veins. A no. 20 cannula may be acceptable for arterial puncture, but a no. 14 catheter is preferable for intravenous and central venous pressure requirements. All transfused blood should be warmed and filtered.

A second use of the plastic cannula is in aspirating the pericardium in cases of shock accompanying a high central venous pressure. Cannulation of the pericardium may be both diagnostic and curative.

Disrupted Viscera

A *Foley catheter* in the bladder provides hourly assessment of renal function and the efficacy of intravenous therapy. It also provides the means of identifying a urinary tract injury, hematuria being the signal. Retrograde cystography should be performed whenever the pelvic bones are fractured. If there is resistance to catheterization, no force should be exerted for the urethra may be ruptured. A small suprapubic polyethylene catheter will then provide temporary decompression of the bladder.

A *nasogastric tube* must be inserted, but the ideal of a totally empty stomach is seldom achieved; the larger nasogastric tube the better. A sump catheter saves frequent irrigations. Blood may be the first clue to visceral injury. A nasogastric tube with a radiopaque marker may occasionally help one to identify an otherwise unsuspected traumatic rupture of the diaphragm. From a therapeutic viewpoint, a functioning nasogastric tube will reduce aspiration hazards and prevent or treat gastric and bowel dilatation.

Placement of a *peritoneal dialysis catheter* allows one to institute lavage of the peritoneum with saline and examination of the recovered fluid—the essential operation in determining intraperitoneal bleeding or visceral tears. Waiting for physical signs to develop and even doing four quadrant taps are, by comparison, entirely inadequate. With the bladder empty, the multiple fenestrated dialysis catheter is inserted through a trocar 5 to 7.5 cm below the navel in the midline. One litre of intravenous isotonic salt solution is instilled, after which the bottle is lowered to the floor. With grossly bloody drainage or the finding of more than 100 000 erythrocytes per millilitre, intra-abdominal bleeding must be assumed and prompt operation is indicated. This test, properly performed, reflects the true condition of intra-abdominal viscera in 96% of patients studied.[5]

Examine Elsewhere

One must consider that widening of the mediastinum may be the sole clue to injury of the thoracic aorta; this is an indication for emergency aortography or transfer to a hospital where this can be done. Remember that the diaphragm is often torn and seldom recognized, whereas the esophagus is seldom torn and frequently considered. Heart valves may be rendered incompetent by a severe trauma. Is a regurgitant murmur present? The thoracic spine is part of the chest; are there any fractures, and are the movement and sensation of the lower extremities normal?

The retroperitoneal portion of the duodenum, vena cava and the pancreas harbour the silent killers of the abdomen. There are clues to each if the right questions are asked.

Finally, the condition of the central nervous system and of the peripheral nerves, bones, arteries, tendons and injuries of the soft tissues must be considered. Except for injuries of the central nervous system, however, these are not usually of first priority.

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FULMINATING NONCLOSTRIDIAL GAS-FORMING INFECTION: A CASE OF NECROTIZING FASCIITIS

R. J. BLANCHARD, MD, FRCS[C]*

Summary: Perianal infection in a 40-year-old man resulted in extensive necrotizing fasciitis of the retroperitoneal space and septic shock. Despite the fact that radiography revealed linear streaking in the belly of the psoas muscle due to gas formation, the nature of the infection was necrotizing fasciitis and not myonecrosis. This contradicts Brightmore's contention that such a finding always indicates clostridial myonecrosis. Despite the absence of *Clostridium welchii*, necrotizing fasciitis is none the less extremely serious, usually occurring in the limbs or abdominal wall superficial to muscle layers. The case reported is unusual in that infection affected the fascia deep to abdominal muscles in the retroperitoneal space, where surgical exposure is difficult. An appropriate surgical approach afforded adequate treatment.

Résumé: Il s'agit d'un cas d'infection péri-anale qui s'est compliqué d'une cellulite nécrosante extensive de l'espace rétropéritonéal et d'un choc septique. Ce cas possède de multiples facettes instructives. Quoique la radiographie de l'abdomen montrât des stries linéaires du muscle psoas contenant des gaz, il s'agissait bien d'une cellulite nécrosante et non d'une myonécrose. Ceci contredit l'affirmation de Brightmore qui veut qu'une telle découverte radiologique signe une myonécrose à Clostridium. Malgré l'absence de *Clostridium welchii*, la cellulite nécrosante demeure une infection très sérieuse. Ce type d'infection survient généralement au niveau des membres et des couches superficielles de l'abdomen. Ce cas est inhabituel du fait que l'infection touchait le fascia profond de l'abdomen dans l'espace rétropéritonéal où l'exposition chirurgicale est très difficile. Nous avons décrit une approche chirurgicale qui semble avoir été, un traitement efficace dans le cas présent.

THE clinical picture of shock associated with infection and gas in tissues indicates an extremely serious disorder regardless of the organism causing the infection. Although radical debridement and adequate drainage are of the utmost importance, the ideal may not always be attainable. The extent of the infection and the identity of the causative organism or organisms must be determined immediately and treatment instituted without delay. One of the features of gas-forming infections by whatever cause is rapid and extensive spread either along fascial planes or along muscle bellies. When infection originates in or enters a body cavity, palpation may not afford adequate information, but radiographic studies may give a reasonable estimation of the extent of infection.

Brightmore[1] has stated that "radiologically it is only in clostridial myonecrosis that soft tissue gas is seen as intramuscular multilinear streaks; in all other infections it is extramuscular, usually as bubbles". Our experience contradicts this statement. A patient recently treated for nonclostridial gas-forming infection illustrates that intramuscular multilinear streaks can be seen in such infections; that perianal disease can be most serious if not treated adequately and promptly; and that necrotizing fasciitis may tax one's ingenuity in treatment.

CASE REPORT

A 40-year-old native Indian man was transferred from a northern hospital with a 1-week history of increasing pain in the perianal region and a recent, painful, tender swelling in the right flank and lower abdomen. He had been treated with antibiotics but not by incision and drainage. He was febrile, hypotensive and drowsy. The left buttock was swollen and red; a patch of skin approximately 5 cm from the anus was necrotic. Crepitus was detected in the left perianal region and, on rectal examination, extreme tenderness to the left was noted. There was generalized swelling of the right lateral abdominal wall and flank with guarding and tenderness also; and crepitus was palpable in the right flank. The remainder of the abdomen was soft and no tenderness of the left side of the abdomen was elicited. The leukocyte count was 11 400/mm^3 and the hemoglobin value, 11.4 g/dl; the plasma glucose concentration was 130 mg/dl.

A radiograph of the abdomen taken with the patient supine showed a large collection of extraluminal gas in both ischiorectal spaces

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and over both levators extending up the right iliacus muscle into the right retroperitoneal space as high as the posterior insertion of the diaphragm; there was, in addition, linear streaking of gas in the right psoas muscle (Fig. 1). Gram's staining of the fluid from the perianal region revealed gram-positive cocci and pleomorphic gram-negative rods. Cultures later revealed the following organisms: *Escherichia coli,* 4+; *Bacteroides* sp., 4+; *Diphtheroides* sp., 4+; anaerobic streptococci, 3+; and *Klebsiella* sp., 4+. The diagnosis of necrotizing fasciitis was made. Samples of blood for culture were drawn which later proved to be sterile, as did the urine.

The patient was treated for shock. Ampicillin (2 g q4h), clindamycin (300 mg q6h) and gentamicin (80 mg q8h adjusted to blood concentrations) were administered intravenously.

On the day of admission a long transverse incision was made in the flank extending from the middle of the right rectus muscle posterolaterally. Gas was found in the rectus sheath and black discolouration was obvious over the muscle fibres of the rectus abdominis; but when the discoloured tissue was scraped the underlying muscle appeared viable and normal. Incision of the muscles of the abdominal wall revealed the plane of spread of the infection to be in the subtransversus abdominis space along the transversalis fascia. This space was opened; the intact peritoneum was retracted medially as in lumbar sympathectomy exposure, and the space was opened from the posterior insertion of the right half of the diaphragm downwards over the right iliacus muscle and posteromedially over the psoas muscle. A large volume of opalescent, extremely foul-smelling fluid was aspirated from this space; large segments of necrotic transversalis fascia were also removed. Cultures of this material grew the following organisms: *E. coli,* 4+, *Bacteroides* sp., 4+; anaerobic streptococci, 4+; and anaerobic diphtheroids, 4+. All of the muscles of the abdominal wall were viable and intact but edematous. There was no evidence of any intraperitoneal communication, and palpation through the peritoneum revealed no evidence of an abscess in the right lower quadrant. Three large sump drains were placed: one superiorly, one medially over the psoas muscle and one inferiorly over the iliacus muscle.

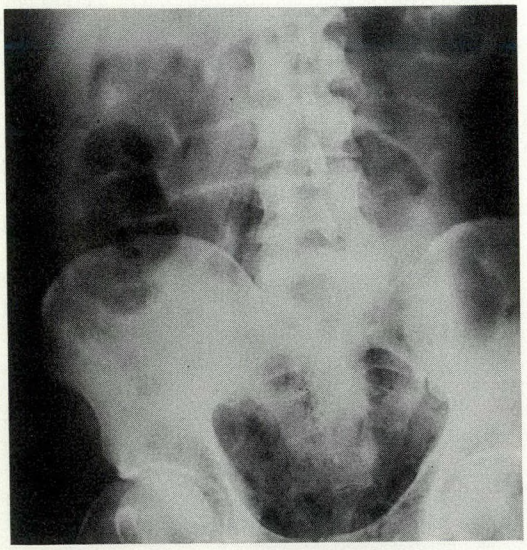

Fig. 1.—Linear streaking in right psoas due to gas, and scattered gas in pelvis and right retroperitoneal space on admission film.

The perineal region was then approached and all necrotic skin excised. A large amount of necrotic fat with multiple serpiginous tracts extended to the gluteus maximus muscle on the left, but the muscle was not infected. The process extended upwards on the left lateral side of the rectum and posteriorly to the rectum. Necrotic tissue and gas were found below and above the left levator muscle, but the levator muscle itself was springy and appeared viable. The necrotic tissue and gas were traced over the right levator and on to the right iliacus muscle. These spaces were all opened and tissue debrided as thoroughly as possible. A large sump drain was inserted above each levator muscle. No source of rectal leak was found at this operation.

In the first 12 hours following admission, the patient's systolic blood pressure never exceeded 80 mm Hg; also there was evidence of severe pulmonary insufficiency. Fluid administration was monitored by means of a Swan-Ganz catheter for measurement of pulmonary artery and wedge pressures. After an endotracheal tube had been inserted a ventilator (positive end-expiratory pressure, 10 cm H_2O) was used to maintain adequate oxygenation.

In the first 24 hours after admission the patient's platelet count fell from 155 000 to 38 000/mm^3. Two days following admission the patient sustained a massive hemorrhage from acute gastritis and reflux esophagitis, diagnosed gastroscopically. This was treated successfully by gastric-balloon cooling for 20 hours. Dopamine hydrochloride was required for the first few days to maintain an adequate blood pressure. An abdominal radiograph at this time showed that the previously

seen bubbles of gas in the soft tissue, and linear streaking in the right psoas muscle had disappeared (Fig. 2).

Further debridement of the perianal region was required on three occasions. On the first occasion a small posterior anal fissure was found to communicate with the debrided region in the ischiorectal fossa; this epithelial defect may have led to the formation of the ischiorectal abscess. The sump drains were removed sequentially and the flank incision was subsequently closed by secondary suturing. The perineal wound was allowed to close by granulation and contraction. There is a persistent ridge of scar posterior to the anus and loss of external and internal sphincter in the posterolateral aspect of the anus. However, the remaining sphincter maintains complete continence and patient has normal, formed stools. The patient returned to work 1 month after discharge and is in good health 14 months after his original admission.

Discussion

Usually perianal infections are localized, but this is not always the case. In reviewing patients with gas gangrene of the scrotum and perineum (Fournier's gangrene), Himal, McLean and Duff[2] found that all 12 cases were associated with inadequately drained perianal infections, and in only two cases were they able to culture *Clostridium welchii*. The present case illustrates a different and very uncommon complication of delayed drainage of an ischiorectal abscess. The infection extended internally along the levator muscles, iliacus muscle, psoas muscle and retroperitoneal space rather than externally into the subcutaneous fascia of the perineum and lower abdominal wall.

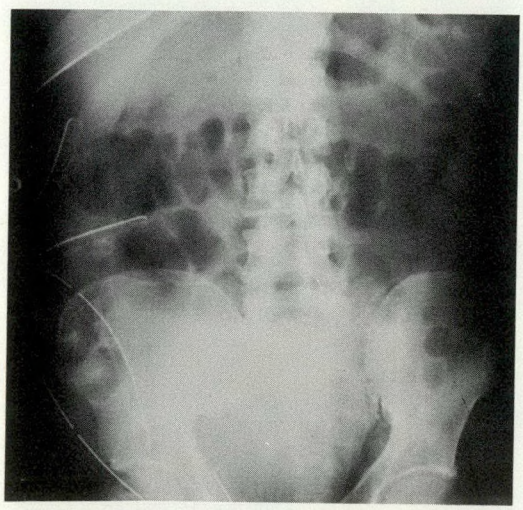

Fig. 2.—Absence of gas in psoas or retroperitoneal space 2 days after admission.

The causative organisms were aerobic gram-negative rods together with anaerobic rods and cocci. The tissues primarily infected were fascia and fat. To avoid the confusion of terms regarding such fascial infections, the term "necrotizing fasciitis" should be used. Although several authors have attempted to define necrotizing fasciitis in relation to the causative organisms, it would seem more appropriate to include under this designation all infections in which fascia is the primary tissue affected, regardless of the actual organism or organisms involved. Rea and Wyrick,[3] in reviewing 44 patients diagnosed as having "necrotizing fasciitis", found a variety of causative organisms and, in some cases, mixtures of organisms. Meade and Mueller[4] have commented on the presence of mixed organisms in two cases of necrotizing infection of the subcutaneous tissues and fascia. Finding two or more species of bacteria in such infections has prompted some clinicians to label the infection "synergistic gangrene";[5, 6] others have labelled similar infections "progressive gangrene".[7] Because some of the organisms that may cause necrosis and infection of fascia produce gas, the terminology is further confused by this additional feature. Brightmore[1] described such a patient as having a "non-clostridial gas infection". Several observers restrict the term "necrotizing fasciitis" to those infections in which only β-hemolytic streptococci are the causative organisms.[8-10] In order to avoid such confusion, necrotizing fasciitis should be defined by the tissue involved and not by the infecting organisms.

The mechanism by which infections resulting from a variety of different organisms may affect fascia primarily, sparing muscle and other tissues, is not entirely clear, but it may be related more to the host than the infecting agent. Whatever the mechanism, infections producing necrosis of the fascia by whatever organism or combinations of organisms can be extremely serious, sometimes leading to amputation or death.

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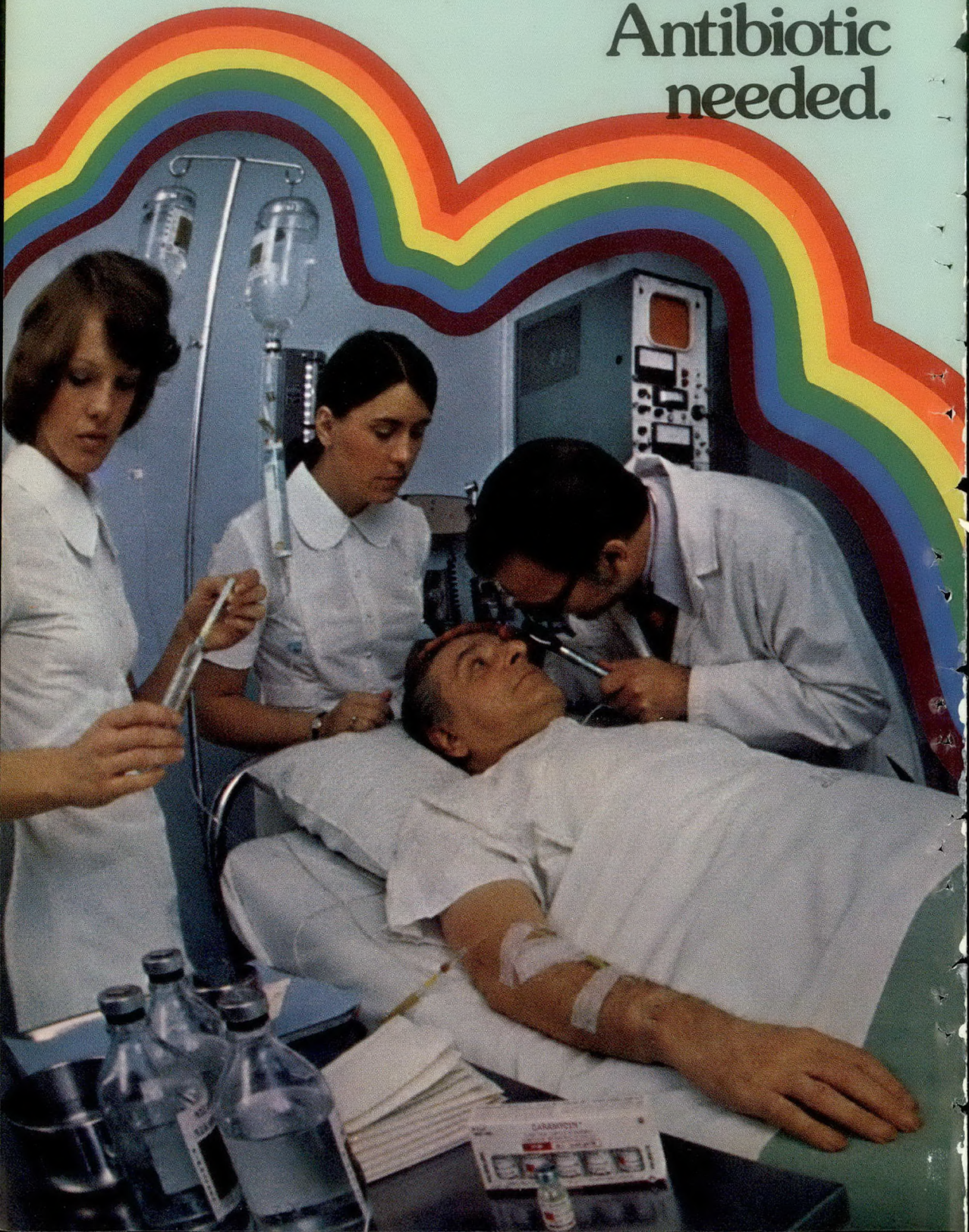

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from a myonecrosis, because the treatment is different. Myonecrosis is extremely rare apart from infection by clostridial organisms. In most clostridial infections, Gram's staining and culture reveal this organism alone. When *Clostridium* spp. are present in combination with large numbers of some other organism or organisms, the infection is usually fascial and not muscular. Hence, it is of the greatest importance to identify the causative organism or organisms immediately. Gram's staining is usually helpful. Cultures must be done for both aerobic and anaerobic organisms. Radiographs revealing gas in tissue are not diagnostic of the type of organisms causing the infection.

It is also essential to emphasize that, if complications are to be avoided, perianal and ischiorectal abscesses must be incised and drained early and completely. Sometimes this requires unroofing by removal of skin edges. One should be prepared to perform exploratory incision in patients with perianal tenderness, malaise, fever and leukocytosis, even in the absence of obvious mass or fluctuation. To wait for fluctuation can be dangerous, especially in cases of deep abscesses.

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DUODENAL OBSTRUCTION RELATED TO BENIGN BILIARY TRACT DISEASE*

D. M. GRACE, MD, FRCS[C]† and H. S. HIMAL, MD, FRCS[C]

Summary: Duodenal obstruction in six patients was related to biliary tract disease and previous abdominal surgery. Causes of obstruction were adhesions to the inflamed gallbladder, adhesions to the gallbladder bed after cholecystectomy, intramural hematoma after transduodenal exploration of the common bile duct and severe pancreatitis after common bile duct exploration. Measures that may possibly prevent duodenal obstruction include early recognition and treatment of cholelithiasis, positioning of omentum between duodenum and gallbladder bed after cholecystectomy, avoidance of transduodenal exploration where possible and careful duodenal closure if necessary, avoidance of forceful dilatation during common bile duct exploration, and a decrease in the number of negative explorations by increased use of cystic duct cholangiography.

Résumé: Chez six malades, nous avons noté une occlusion duodénale qui provenait de pathologies des voies biliaires et d'opérations abdominales antérieures. Parmi les causes de l'obstruction figuraient: des adhérences à une vésicule biliaire enflammée, des adhérences au lit de la vésicule après cholécystectomie, un hématome intramural consécutif à une exploration du cholédoque par voie transduodénale et une pancréatite sévère après exploration du dit canal biliaire. Parmi les mesures qui seraient susceptibles de prévenir l'obstruction duodénale, on peut citer le diagnostic et le traitement précoces de la cholélithiase, la mise en place de l'épiploon entre le duodénum et le lit de la vésicule biliaire après cholécystectomie, éviter, dans la mesure du possible, l'exploration transduodénale et fermer soigneusement le duodénum en cas de besoin, éviter aussi la dilatation forcée durant l'exploration du cholédoque et tenter de réduire le nombre d'explorations négatives en se servant davantage de la cholangiographie du canal cystique.

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BILIARY tract disease may produce duodenal obstruction in a variety of ways, and this problem may not be recognized by individual surgeons more familiar with cases of pyloric obstruction due to peptic ulceration or the occasional case of malignant invasion of the duodenum. In this paper we describe some of the ways in which biliary tract disease may produce duodenal obstruction, either by preoperative adhesions or postoperative complications, and emphasize some preventive measures. Six cases of duodenal obstruction form the basis of this report.

CASE REPORTS

Case 1.—A 73-year-old woman was admitted with epigastric pain and vomiting. She knew she had gallstones but had refused operation 2 years before. An upper gastrointestinal series showed obstruction of the proximal duodenum. Laparotomy revealed obstruction of the duodenum by dense adhesions to the gallbladder bed. Because it was thought that the massive adhesions would make cholecystectomy dangerous, a gastrojejunostomy was performed. Recovery was uneventful. A follow-up barium study 15 months later showed that the gastroenterostomy was patent. Little barium passed the duodenum and there was air in the biliary tree.

Case 2.—A 45-year-old man was admitted because of recurrent upper abdominal pain and vomiting. Radiographs of the upper gastrointestinal tract showed an irregular antrum and a narrow duodenal bulb. A duodenal ulcer had been diagnosed 10 years before, gastric acid secretion being elevated at that time. Gastroscopy showed no abnormality. Laparotomy disclosed massive adhesions between the chronically inflamed gallbladder and the proximal duodenum causing extrinsic obstruction. There was no evidence for a duodenal ulcer. The gallbladder was removed and the postoperative course was uneventful.

Case 3.—A 34-year-old woman underwent cholecystectomy, and during the procedure a single stone was found in Hartmann's pouch. The patient ran a low-grade fever (to 37.8°C) for a few days but otherwise did well and was

discharged 8 days after operation. One month after the first admission she was readmitted because of recurrent nausea and vomiting. Upper gastrointestinal radiographs showed extrinsic narrowing of the postbulbar duodenum. The mucosa was intact. The narrowing was thought to be the result of hematoma formation, and the patient was treated with intravenous fluids and nasogastric suction. Her condition improved and she was discharged.

She was admitted for the third time approximately 7 weeks after her first admission. She had severe nausea and vomiting. An upper gastrointestinal radiographic series showed a grossly dilated fluid-filled stomach with obstruction in the first part of the duodenum. Laparotomy revealed gross scarring and adhesions about the first and second portions of the duodenum. The duodenum was adherent to the gallbladder bed. The duodenum was freed and the narrowed portion dilated through a gastrotomy incision with Hegar dilators. Cultures of material taken from this area were negative, but wound infection developed after operation. Recovery was otherwise uncomplicated. A further barium study showed prompt gastric emptying with only mild extrinsic compression of the duodenal bulb. She has remained well.

Case 4.—A 43-year-old woman had a history of upper abdominal pain and radiographically proven gallstones. Cholecystectomy and hiatus hernia repair were performed. No abnormalities were noted. The gallbladder fossa was not oversewn. The postoperative course was complicated by upper abdominal pain and vomiting, and a nasogastric tube was inserted. Nasogastric suction yielded a large amount of fluid, and a barium meal confirmed obstruction in the proximal duodenum rather than failure of gastric emptying.

Laparotomy revealed dense adhesions between the gallbladder bed and the first and second portions of the duodenum. No local abscess or acute inflammation was found. Some of the adhesions were divided and a gastrojejunostomy was carried out. A wound infection developed but ultimately the patient did well.

Case 5.—A 36-year-old woman had recurrent attacks of right upper quadrant pain, several of which were acute. Laparotomy revealed four stones in the gallbladder, one of the stones being quite small. The common duct was dilated and explored; no stones were found, but on cholangiography no dye entered the duodenum. The duodenum was opened through a transverse incision and a tight papilla of Vater dilated from below. No stones were found. The duodenum was closed in two layers.

The postoperative course was complicated by fever and melena. Her hematocrit was 19%; her pulse rate, 130 beats/min; her blood pressure, 90/60 mm Hg; and nasogastric suction returned "coffee-ground" material. Two units of blood were given. A barium study 4 days later showed a 3-cm intramural defect in the second portion of the duodenum with narrowing of the lumen. The lesion was thought to be a hematoma. Gastric suction was necessary for 10 days (average 24-hour volume, 1 *l*). She was eventually discharged in good condition.

Case 6.—A 61-year-old woman had had a cholecystectomy 3 years before the present admission but had complained of persistent epigastric pain since then. An upper gastrointestinal series showed a hiatus hernia without reflux. An intravenous cholangiogram revealed that the common duct was 14 mm in calibre and that it contained no stones but that it tapered abruptly in the distal portion. The serum alkaline phosphatase value was elevated (28 King-Armstrong units; normal, to 13 KA units) and the serum amylase value was normal.

The common duct was then explored. The duct was indeed dilated, but no stones were found. Bakes' dilators (size, up to 7 mm) were passed through the distal end of the duct. The postoperative course was complicated by abdominal pain and fever, calf-vein thrombophlebitis and urinary tract infection.

Two weeks after operation her abdomen became distended. Gastric suction yielded 2000 ml over 24 hours. An upper gastrointestinal series demonstrated virtually complete obstruction of the second portion of the duodenum. An inflammatory mass distended the duodenal loop, and irregularity of the third portion of the duodenum was visualized; the diagnosis of pancreatitis was suggested. Gastric suction, which was necessary intermittently for approximately 1 month, continued to yield and average of 2 *l*/d, and vomiting followed clamping of the tube. A second laparotomy showed that the pancreas was firm and that there were adjacent areas of fat necrosis and adhesions to the gallbladder bed. A gastrojejunostomy was performed. Return of bowel function was slow but after this recovery was uneventful.

The clinical and pathologic features of these six cases, and their treatment are summarized in Table I.

TABLE I.—SUMMARY OF CLINICAL AND PATHOLOGIC FEATURES AND TREATMENT OF SIX CASES OF DUODENAL OBSTRUCTION

Case no.	*Sex and age (yr)*	*Cause of obstruction*	*Treatment*	*Comment*
1	F, 73	Adhesions to chronically inflamed gallbladder	Gastrojejunostomy	Gallbladder not resected because of adhesions; air in biliary tree
2	M, 45	Adhesions to gallbladder	Cholecystectomy	Uncomplicated recovery
3	F, 34	Adhesions to gallbladder bed 1 mo after cholecystectomy	Freeing of duodenum; dilatation with Hegar dilators	Wound infection; excellent recovery
4	F, 43	Adhesions to gallbladder bed 2 wk after cholecystectomy	Gastrojejunostomy	Wound infection; otherwise did well
5	F, 36	Intramural hematoma after transduodenal exploration	Nasogastric suction (10 d)	GI hemorrhage; required 2 units of blood
6	F, 61	Pancreatitis after exploration of common duct	Nasogastric suction (approx. 1 mo); gastrojejunostomy	Negative results of exploration of common duct

DISCUSSION

The many causes of duodenal obstruction are summarized in Table II. The only common cause, however, is peptic ulceration of the pylorus and proximal duodenum; tumour invasion, the second ranking cause, is much less common. With respect to symptoms, nausea and frequent vomiting, with or without bile depending on the level of obstruction, are not unusual. Nevertheless, duodenal obstruction may not be recognized initially in a patient who is known to have gallstones or who has undergone an abdominal operation.

In relation to the gallbladder, the most common cause of duodenal obstruction is a gallstone. No such examples have been seen at our hospital. Impaction of a gallstone is, of course, most common in the distal ileum but it can occur anywhere from the stomach to the rectum. Several examples of this type of duodenal obstruction have been reported.[1-4] Adhesions between the inflamed gallbladder and the adjacent duodenum are not unusual, and radiologists occasionally

TABLE II.—CAUSES OF DUODENAL OBSTRUCTION*

	Acquired		
Congenital	*Extramural*	*Intramural*	*Intraluminal*
Arteriomesenteric Peritoneal bands Abnormal midgut rotation Cysts and duplications Atresia Annular pancreas Duodenal ptosis, extreme	Adhesions Stomach, gallbladder, pancreas Tumours Stomach) implantation or Pancreas) direct invasion Colon, etc.) Primary retroperitoneal Lymph node: lymphoma etc. Cysts Pancreas, kidney echinococcal Vascular disorder Aortic aneurysm, Superior mesenteric artery	Tumours Diverticuli Peptic ulceration Inflammatory disorder Tuberculosis, syphilis Crohn's disease Hematoma formation Anticoagulants, trauma, operation	Bezoar Foreign body Gallstone Parasites

*Modified after Bockus HL: *Gastroenterology*, v 2, second ed, Philadelphia, Saunders, 1964, p 102.

describe a gallbladder impression on the proximal duodenum. Inflammation may progress to abscess formation between gallbladder and duodenum. This may by itself cause duodenal obstruction[5] or form a fistula. One of our patients with known gallstones (case 1) had refused operation, and duodenal obstruction subsequently required gastrojejunostomy. In another (case 2) duodenal obstruction was thought to be due to peptic ulceration, but the radiographic appearance was not completely typical; obstruction actually resulted from adhesions to an inflamed gallbladder, and cholecystectomy was curative. Early cholecystectomy would, of course, prevent this disorder.

Some degree of adhesion formation to the gallbladder bed after cholecystectomy is not uncommon, and there is no easy way to identify those patients in whom adhesions will form. Some surgeons close the peritoneum of the gallbladder bed and others do not, but this may not be an important factor. Ellis[6] stated that adhesions form in areas of ischemic tissue and not in areas devoid of peritoneum. He found more adhesions to forcibly closed areas of peritoneum than to large deperitonealized areas allowed to heal on their own. In our cases 3 and 4, the gallbladder bed was left open; in neither was there clear evidence of infection, although this may have been a factor because wound infection after relief of duodenal obstruction developed in both. Because adhesions are difficult to predict and prevent, it seems wise to avoid contact between the duodenum and gallbladder bed. This is best accomplished by placing the omentum when available between the gallbladder bed and duodenum after cholecystectomy. This will prevent the duodenum from being involved in an inflammatory process.

Intramural hematoma of the duodenum is a relatively rare result of blunt abdominal trauma; it occurs especially in children,[7] and also in patients being treated with anticoagulants.[8] Duodenal obstruction may be partial or complete. Treatment has been either conservative (administration of intravenous fluids and nasogastric suction) or operative (evacuation of the hematoma and occasionally gastroenterostomy). Any part of the intestine opened at operation may be the site of intraluminal or intramural bleeding and the duodenum is no exception. In our case 5 the patient sustained an upper gastrointestinal hemorrhage requiring transfusion after transduodenal exploration of the common bile duct. Signs of duodenal obstruction then developed, with an intramural filling defect visible on barium study. The appearance was not the classical "coil spring" described by Felson and Levin.[9] Conservative treatment with intravenous fluids and prolonged nasogastric suction was successful. The best treatment is, of course, prevention; one must avoid opening the duodenum when possible and observe careful hemostasis.

Pancreatitis is a rare but lethal postoperative complication after any upper abdominal operation, but especially common bile duct exploration.[10] Recognition is late and the mortality is high. Duodenal obstruction is rare but it may follow paralytic ileus.[11] Surgical bypass is necessary if obstruction persists and either gastrojejunostomy or duodenojejunostomy may be performed. Biliary decompression may also be necessary in some cases. In our case 6 pancreatitis was not recognized until signs of duodenal obstruction became evident. Pancreatitis may have been caused by manipulation of the pancreas or by forcible dilatation of the distal duct with Bakes' dilators. We now use dilators only if rubber catheters will not pass the distal end of the duct. In our patient, conservative treatment with gastric suction and intravenous fluids was carried on intermittently for nearly 1 month. As a result, we would now recommend earlier bypass if duodenal obstruction complicates pancreatitis.

The type of decompression for duodenal obstruction is still a matter of debate. Probably a duodenojejunostomy should be performed if this is possible, especially for distal obstruction.[12] In our three cases treated by bypass (cases 1, 4 and 6), inflammation around the proximal duodenum was such that gastrojejunostomy was necessary; all patients did well. Although this procedure is ulcerogenic in patients with preexisting peptic ulceration, it seems to be well tolerated by those with benign or malignant obstruction of the proximal duodenum. These cases show that the duodenum may be obstructed by preoperative adhe-

sions to the gallbladder or by postoperative complications. Recognition allows treatment by cholecystectomy or gastrojejunostomy. The best treatment, however, is prevention. Gallbladders containing stones should be removed whenever possible before inflammation progresses and leads to adhesions and obstruction. Positioning of the omentum between the duodenum and liver bed after cholecystectomy prevents the duodenum from being drawn upward into an inflammatory process. Careful duodenal closure and avoidance of unnecessary duodenotomy will help prevent duodenal hematomas. Pancreatitis following common duct exploration may be rare if forceful dilatation is avoided and if the number of explorations is decreased by cystic duct cholangiography.

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SPONTANEOUS RESOLUTION OF A PANCREATIC PSEUDOCYST BY PERFORATION INTO THE COLON

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Summary: Spontaneous perforation of a pancreatic pseudocyst into the colon, diagnosed radiologically, resulted in spontaneous cure. Disappearance of a previously palpable abdominal mass suggested perforation of the cyst into the stomach or colon. Surgical treatment was not required. Such an occurrence has not been reported previously; it opens to doubt dicta regarding treatment of this rare complication of pancreatic pseudocyst by immediate colostomy.

Résumé: La perforation spontanée d'un pseudokyste pancréatique dans le côlon, qui avait été découvert par radiologie, a guéri spontanément. La disparition d'une masse abdominale qui était auparavant palpable permet de croire à la perforation du kyste dans l'estomac ou dans le côlon. Il n'a pas été nécessaire d'opérer. On n'avait pas encore signalé une telle occurrence et cela pourrait jeter un doute sur l'opinion qu'on doit pratiquer une colostomie immédiate pour traiter cette rare complication d'un pseudokyste pancréatique.

THE details of the natural history of untreated pseudocysts of the pancreas remain unknown. Spontaneous rupture is probably the most common serious complication. Hanna,[1] who collected 45 cases in a review of the world literature up to 1960, estimated that rupture occurs in less than 5% of cases and that it carries a mortality of 50%. Because of an increased tendency towards hemorrhage and sepsis, the colon is considered to be the most dangerous site of spontaneous perforation.[2] Shatney and Sosin[3] have recommended immediate proximal colostomy to reduce fecal contamination of the pseudocyst and its potentially lethal complications. This paper reports a case of spontaneous rupture of pseudocyst into colon, recognized and successfully treated without operation.

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Case Report

A 47-year-old white man was admitted to hospital with epigastric pain, hematemesis and melena. He had been well until the day before admission, when he noted the onset of continuous, dull, epigastric pain. He felt nauseated and vomited three times, bringing up a mouthful of fresh blood on each occasion. Shortly before arriving at the hospital he passed a melena stool. His past history indicated that he often consumed an excess of alcohol. Two years before the present episode he was hospitalized with upper abdominal and back pain; the probable diagnosis was acute pancreatitis, although the serum amylase concentration was normal. Radiographs of the upper and lower gastrointestinal tract and a cholecystogram were also normal.

He was of average stature and nutritional state, and in no distress. The pulse, temperature and blood pressure were normal. The abdomen was slightly distended, and a firm, tender mass (dimensions, approximately 9 x 9 cm) was palpable in the left upper quadrant. The mass was dull on percussion. A firm liver edge was felt about 3 cm below the right costal margin. Rectal examination showed a small quantity of black stool.

The aspirate from nasogastric tube was clear. Laboratory investigations were as follows: hemoglobin concentration, 12.7 g/dl; leukocyte count, 13 400/mm^3 (75% of the cells being polymorphonuclear leukocytes); total serum bilirubin value, 2.5 mg/dl; serum alkaline phosphatase value, 66 IU; serum lactic dehydrogenase, 70 IU, and serum glutamic oxaloacetic transaminase, 51 IU; blood sugar and blood urea nitrogen values, normal; serum electrolyte and serum amylase values, normal; prothrombin time and partial thromboplastin time, normal; urinalysis and 24-hour urinary amylase value, normal.

Esophagogastroscopy on the same day revealed no lesion in either esophagus or stomach. Two units of whole blood were transfused. Radiographs of the upper gastrointestinal tract done on the next day visualized an air-filled mass in the left upper quadrant behind the stomach; the appearance was reported as being "consistent with a pseudocyst of pancreas" (Fig. 1) and a delayed film showed that some contrast material from the transverse colon had entered the cyst (Fig. 2). But now, when the abdomen was re-examined, the mass was no longer palpable. It was recognized that perforation of a colonic or gastric lesion into the lesser sac could present a similar radiographic picture, but the disappearance of the previously palpable mass strongly favoured the diagnosis of a pancreatic pseudocyst with perforation into the stomach or colon.

For the next 6 days the patient had a fever that ranged between 37 and 40°C. Blood, urine and sputum were cultured, but no pathogenic organisms were isolated. Barium enema examination demonstrated a communication between transverse colon and the cyst (Fig. 3). Because of the danger of impending septicemia from colonic contamination a combination of gentamicin, tetracycline and lincomycin was administered. After 24 hours of antibiotic therapy, the temperature returned to normal.

Oral feeding was resumed. A cholecystogram taken before discharge did not show any evidence of gallbladder disease. The patient left hospital on the 20th hospital day. Subsequent

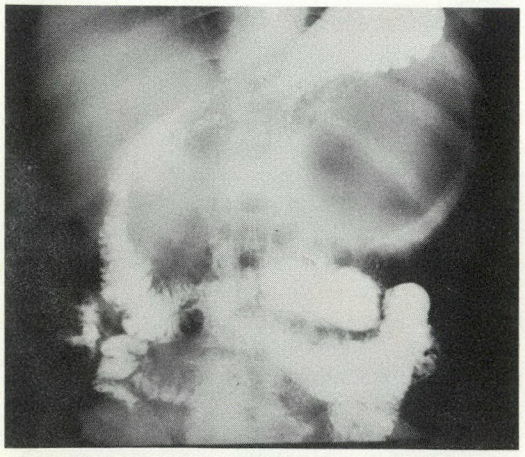

Fig. 1.—Upper gastrointestinal radiograph showing air-filled cyst in left upper quadrant.

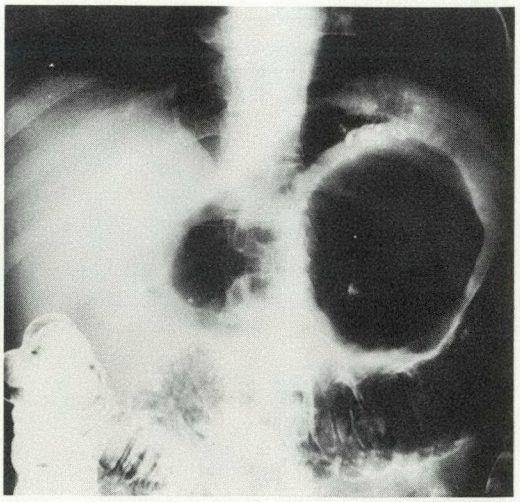

Fig. 2.—Delayed film revealing contrast material in transverse colon and cyst.

barium studies of the upper gastrointestinal tract did not show any evidence of cyst (Fig. 4). When seen 3 months after discharge the patient was free of symptoms and had no evidence of cyst. A barium enema examination was recommended, but the patient declined. When seen again after 8 months he had no abdominal pain and no mass was palpable.

Discussion

The incidence of pseudocyst formation in patients with pancreatitis is about 2%.[4] In less than 5% of cases pseudocysts rupture spontaneously; cysts may rupture into the free peritoneal cavity,[1, 4, 5] stomach and duodenum,[6-8] the colon,[2, 3, 9] through the abdominal wall,[7] and into the pleural cavity.[10]

The mortality in cases of rupture into the peritoneal cavity is higher than that associated with perforation into an abdominal viscus. Hanna[1] estimated that the mortality associated with intraperitoneal rupture was 70%, and others have reported a similar rate in smaller series. Rosenberg, Kahn and Walt,[4] however, have reported 100% survival in four patients.

With respect to spontaneous perforation of pseudocysts into the gastrointestinal tract, current mortality is not known. Perforation into the upper gastrointestinal tract seems to have a better prognosis than perforation into the lower part of the tract. Probably spontaneous perforation into the upper gastrointestinal tract is followed by spontaneous remission of symptoms, so that such cases remain unreported. Perforation into the colon has been considered the most dangerous complication because of the high incidence of fulminating sepsis, hemorrhage or rupture into the peritoneal cavity. Berne and Edmondson,[2] in their review of the literature, found reports of 9 deaths among 14 patients in whom colonic fistula was associated with a pancreatic pseudocyst. They reported on four patients of their own, all of whom died from hemorrhage, one before and three after surgery.

The treatment of spontaneous perforation of pseudocysts into the gastrointestinal tract has not been clearly outlined. Shatney and Sosin[3] suggested that most cases of perforation into the stomach or small bowel can be managed without operation, provided the spontaneous communication is large enough to allow complete drainage of the pseudocyst. If subsequent progress indicates no improvement, internal drainage creating an opening with a diameter of at least 2 cm is indicated. Perforation into the colon, however, is considered a different matter. On the basis of the experience of Berne and Edmondson,[2] several authors have recommended immediate diverting colostomy,

Fig. 3.—Postevacuation radiograph indicating cyst outlined by barium in left upper quadrant.

Fig. 4.—Upper gastrointestinal radiograph, taken 10 days later, shows no sign of cyst.

with or without external drainage of the cyst. Goldin and Wald[9] reported a successful outcome in one patient who was treated in this manner. Cosio and Onstad[11] reported successful treatment of a patient by subtotal colectomy, colostomy and external drainage; the pseudocyst had also communicated with the duodenum. Shatney and Sosin[3] reported on another patient in whom the pseudocyst communicated both with the duodenum and colon. They managed the condition by immediate diverting colostomy, though a fluid-filled cyst could not be demonstrated at operation. In all these reports sepsis was the only complication of spontaneous perforation; this perhaps could have been controlled without operation, as in our case.

Although the risk of sepsis in pseudocystocolostomy has been emphasized, hemorrhage has consistently been reported to be the chief cause of death. Among the 14 patients with colonic fistulas whose cases were reviewed by Berne and Edmondson,[2] 2 had died from sepsis and 7 had died from hemorrhage; moreover, all 4 of their own patients with fistulas into the colon died from hemorrhage. Greenstein, DeMaio and Nabseth[12] have emphasized the seriousness of hemorrhage from pancreatic pseudocysts and have shown that it is often a result of erosion of a large blood vessel. In their experience, massive hemorrhage occurred both from cystocolic and cystogastric fistulas, and could only be controlled by direct ligature of the involved blood vessel. The value of diverting colostomy in preventing hemorrhage is open to question.

The limited experience reported in the literature does not permit one to be dogmatic in outlining the treatment of spontaneous perforation of a pseudocyst into the colon. The precise form of treatment should depend on the clinical situation. If perforation is accompanied by serious hemorrhage, identification of the bleeding by early angiography is indicated. When a large bleeding vessel is detected, an early operation to provide direct ligature of the vessel and possibly drainage of the cyst is required. In the absence of serious hemorrhage, vigorous antibiotic therapy against colonic bacterial infection should be started and continued if there is definite evidence of improvement; complete resolution may ensue, as it did in the present case. However, if sepsis continues and threatens life, external drainage of the pseudocyst is indicated. The value of diverting colostomy in this situation is questionable.

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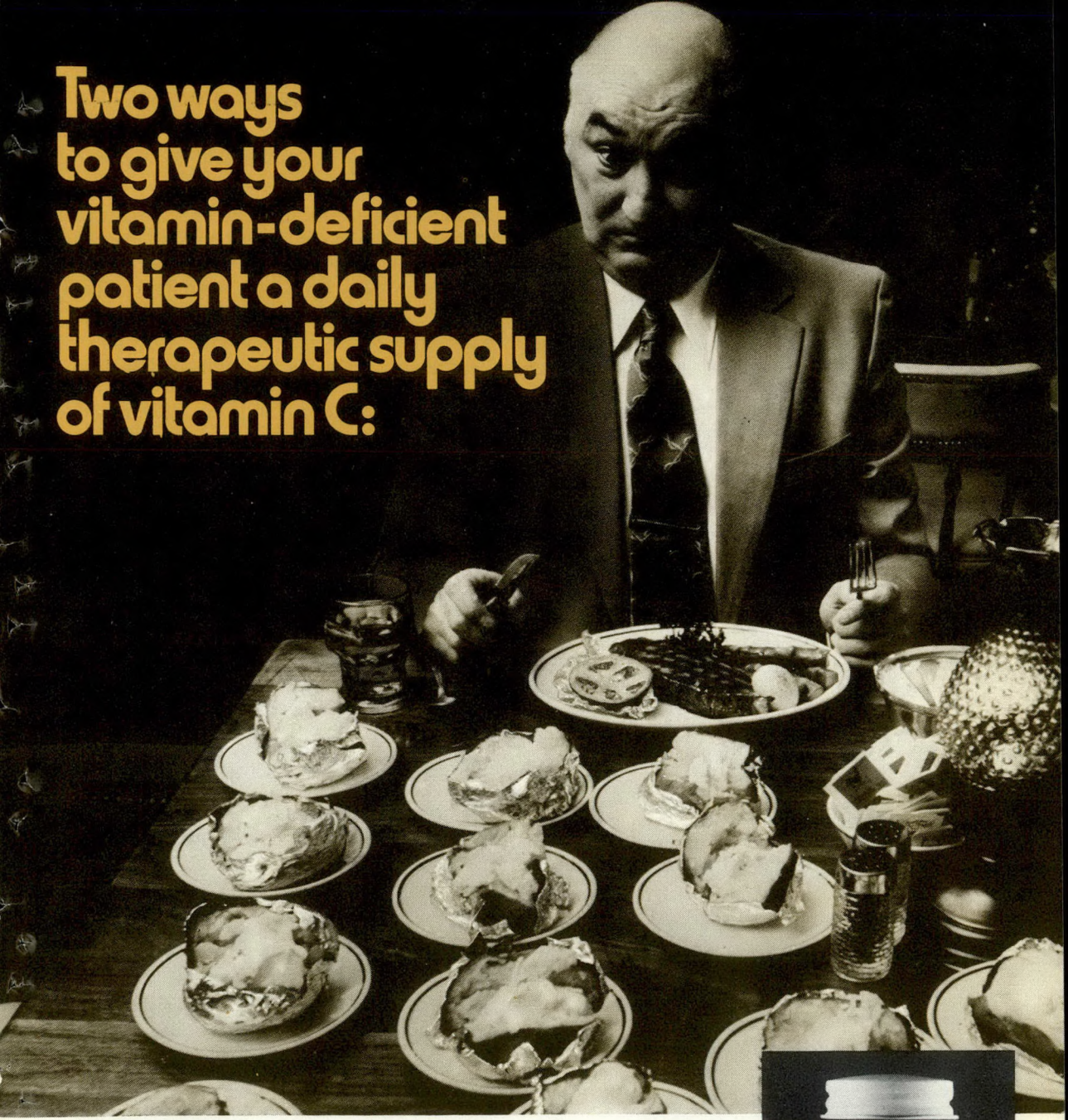

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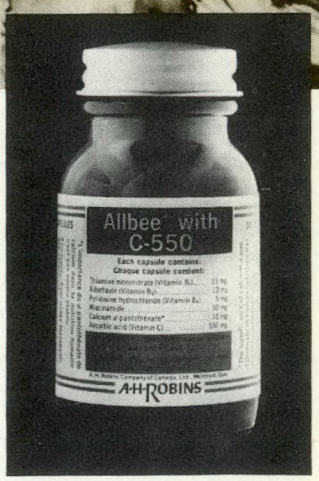

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MALIGNANT DEGENERATION OF A CHONDROMYXOID FIBROMA IN A CHILD*

S. SEHAYIK, MD and M. A. ROSMAN, MD, FRCS[C]

Summary: A 10-year-old boy was treated for a chondromyxoid fibroma of the left femur by curettage, and 2 months later complete healing was demonstrated radiographically. Three years after operation, radiographs revealed replacement of the left ilium, ischium and upper femur, and destruction of the left hip by a soft-tissue and bony mass; a left hemipelvectomy was performed for what was shown to be a chondrosarcoma. Follow-up 10 years after the initial operation and 7 years after the hemipelvectomy has confirmed uneventful recovery in this rare case of malignant degeneration of a proven chondromyxoid fibroma of bone.

Résumé: Nous avons traité par curetage un fibrome chondro-myxoïde du fémur gauche dont souffrait un enfant de 10 ans. Deux mois plus tard, l'examen radiologique montrait une guérison complète. Or, 3 ans après l'opération, la radiographie révélait le remplacement de l'iléon, de l'ischion et de la tête fémorale gauche et la destruction de la hanche gauche par une masse formée de tissu mou et d'os. Nous avons alors pratiqué une hémipelvectomie de ce côté pour traiter ce qui était, en fait, un chondrosarcome. Le malade a été suivi pendant 10 ans après l'opération initiale et 7 ans après l'hémipelvectomie ce qui nous a permis de conclure à la guérison sans incident de ce rare cas de dégénérescence maligne d'un fibrome chondro-myxoïde de l'os.

CHONDROMYXOID fibroma is a rare benign tumour of bone, first described by Jaffe and Lichtenstein[1] in 1948. Most commonly affecting adolescents, it usually develops in the metaphyseal portion of long bones; it most frequently is found in the lower extremities, specifically in the tibia. Complaints of local pain and sometimes a palpable mass are the usual presenting symptoms. The radiographic appearance is that of an eccentric, radiolucent area, distinctly metaphyseal, with cortical expansion and thinning.

Iwata and Coley[2] reported six cases of chondromyxoid fibroma; in one there was secondary malignant degeneration. Aegerter and Kirkpatrick[3] reported a series of 21, in 8 of which there was sarcomatous degeneration. Dahlin[4] stated that he had not seen malignant transformation of a chondromyxoid fibroma, but he emphasized that errors have been made with inadequate diagnosis and treatment of a chondrosarcoma. Jaffe[5] reported one case of malignant change in a chondromyxoid fibroma, and Woods[6] referred only to the fact that malignant transformation does occur.

This paper is a report of a case of chondrosarcomatous degeneration in a proven chondromyxoid fibroma, with a 10-year follow-up.

CASE REPORT

A 10-year-old boy was first seen in 1964, with complaints of left hip pain and limp for 5 months.

Physical examination revealed tenderness along the anterior aspect of the left hip and upper femur, with restricted internal rotation. Radiographs of the left hip demonstrated an area of erosion along the medial border of the femoral neck, with some evidence of new

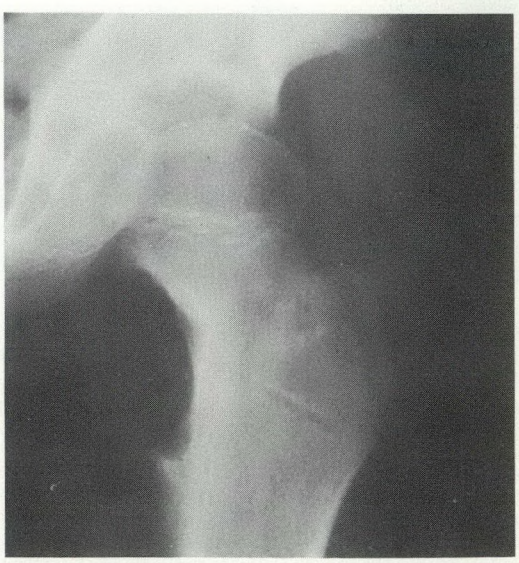

Fig. 1.—Scalloped radiolucent appearance of chondromyxoid fibroma in region of inferior neck of left femur.

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bone formation, and an adjacent soft-tissue mass (length, approximately 4 cm) (Fig. 1). Skeletal survey was normal, as were laboratory findings. The lesion was biopsied and then excised by curettage; the tissue was gelatinous. The pathological diagnosis was chondromyxoid fibroma (Fig. 2). A spica was applied. Two months later he was well and the leg was mobilized; radiographs showed complete healing.

He did well until late 1967, when he complained of insidious onset of pain in his left hip. The hip was painful and motion was greatly restricted. The muscles about the hip were atrophied. Radiographs of the left hip (Fig. 3) revealed replacement of the ilium, ischium and upper femur by a large soft-tissue and bony mass, with expansion and destruction of bone on both sides of the hip joint. Results of skeletal survey, barium enema studies, intravenous pyelography and lymphography were negative. Left femoral arteriography demonstrated increased vascularity in the region of the left hip with stretching of the vessels (Fig. 4).

Microscopic examination of tissue taken by open biopsy revealed chondrosarcoma (Fig. 5). A left hemipelvectomy was later performed. The patient has remained well since last seen in 1974, at age 20 years; the radiographic appearance is shown in Fig. 6.

Discussion

Histologically, chondromyxoid fibroma consists of lobulated areas of myxomatous tissue separated by fibrocellular bands with little tendency for chondroid formation. Many histologic variations of this pattern occur, so that a more confusing or serious microscopic picture results. Differentiation from benign chondroblastoma and malignant chondrosarcoma is more difficult. The clinical and radiologic aspects help to differentiate the various lesions.

In the various reports of the malignant nature of chondromyxoid fibroma, there have been few documented cases[2, 5] of chondrosarcomatous degeneration of a proven chondromyxoid fibroma. Rahimi and colleagues,[7] in an extensive review, stated that no case of malignant change has been convincingly documented, and that all evidence indicates that the risk of malignant transformation of *bona fide* chondromyxoid fibroma, unless radiation is used, is slight. Schajowicz and Gallardo[8] indicated that malignant transformation appears to be exceptional; they had no cases in their series of 32 chondromyxoid fibromas.

Fig. 2.—Histologic features of chondromyxoid fibroma, taken at biopsy from inferior neck of left femur. Note typical lobulated pattern with myxoid matrix and loosely dispersed spindle-shaped cells (hematoxylin and eosin).

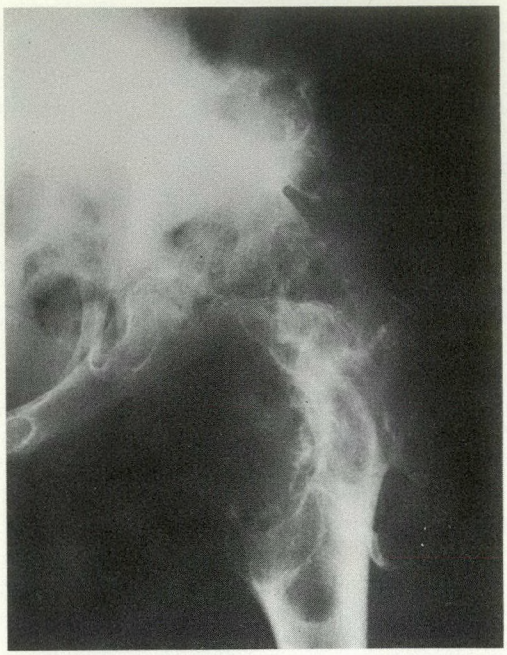


Fig. 3.—Destruction of left hip region 3 years after initial biopsy.

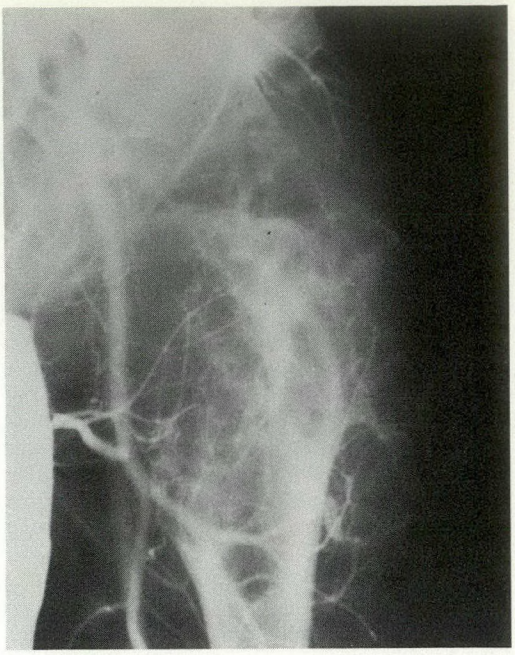


Fig. 4.—Left femoral arteriogram showing extent of disease of left hip.

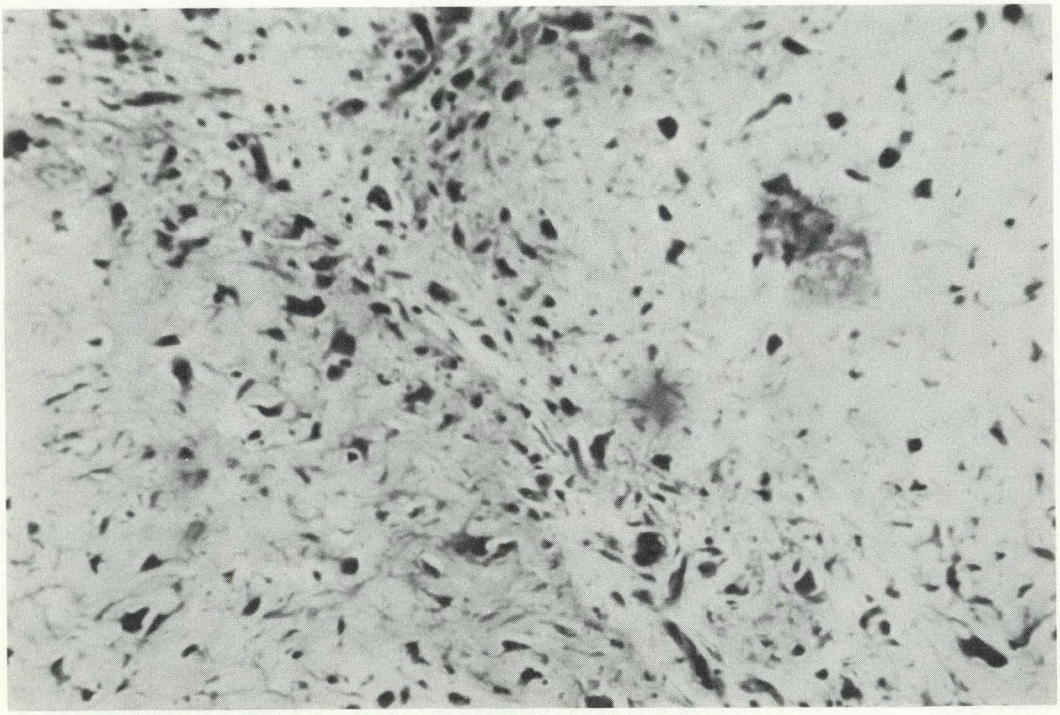


Fig. 5a

Fig. 5.—Sarcomatous degeneration of chondromyxoid fibroma. (a) From amputation specimen. In this field lobulated chondromyxoid pattern is maintained; cells, however, are much larger with irregular hyperchromatic nuclei (hematoxylin and eosin). (b) Another field from amputation specimen. Tumour is frankly malignant and chondrosarcomatous (hematoxylin and eosin). (c) Invasion of subcutaneous tissue overlying recurrent tumour. Diffuse infiltration by malignant cells exhibiting "indian file" pattern (hematoxylin and eosin).

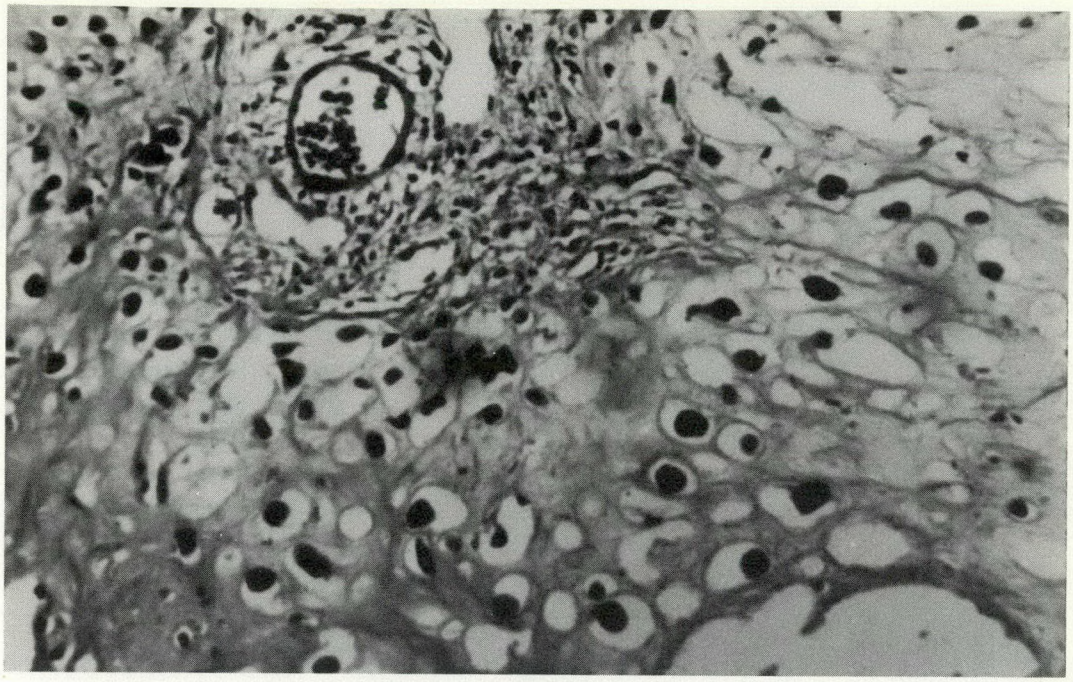


Fig. 5b

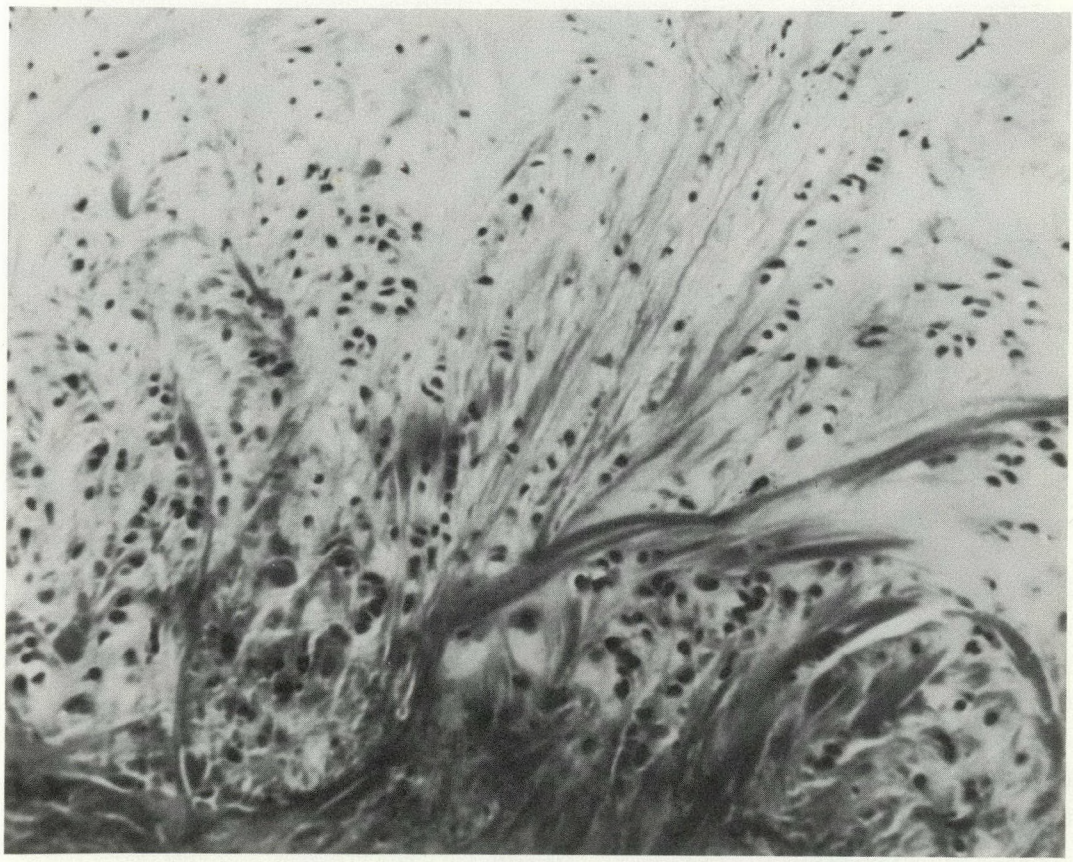
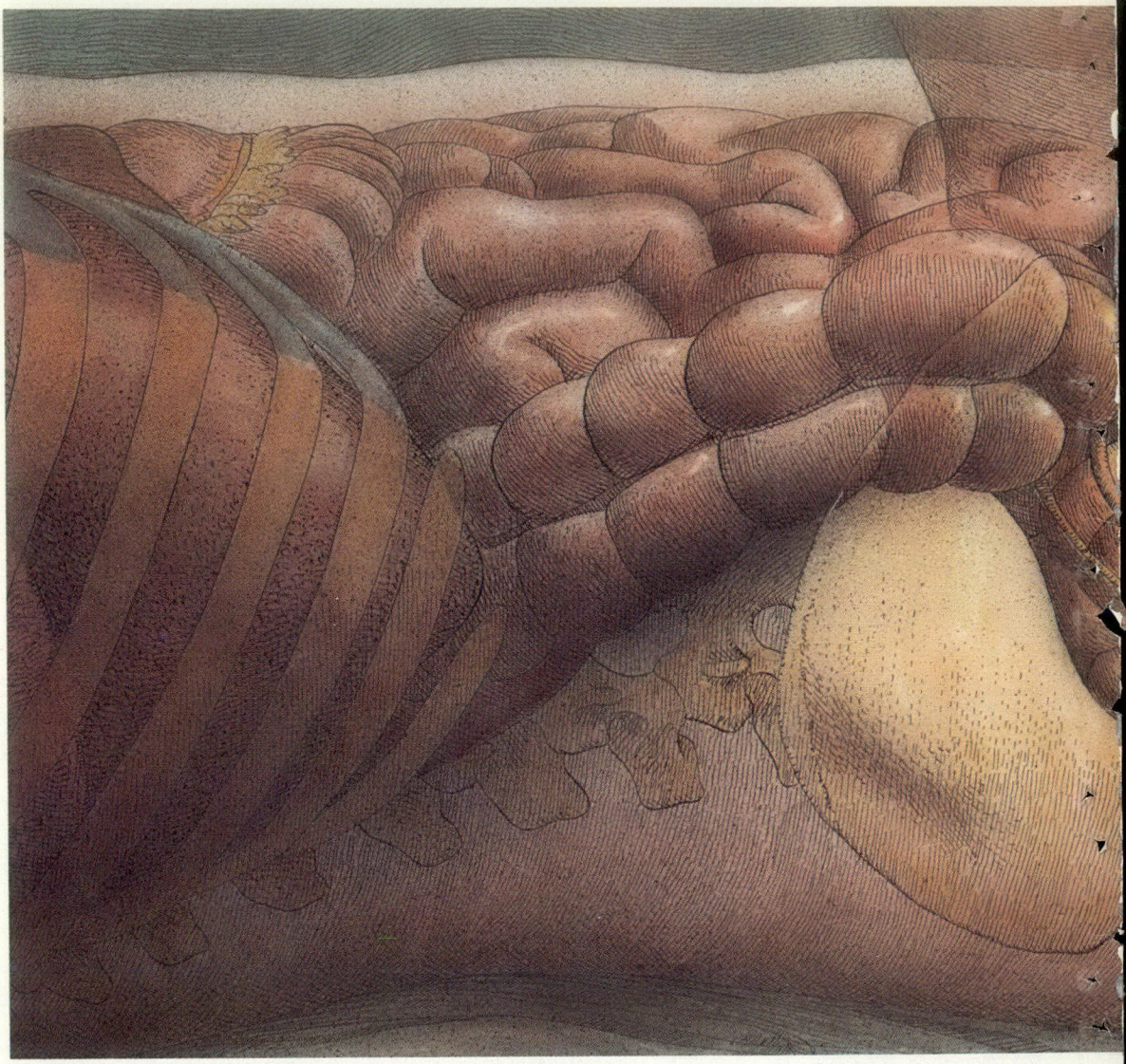


Fig. 5c



an emerging problem.

"Bacteroides species are often overlooked as a cause of serious infection both by clinicians and microbiologists. They are most commonly associated with intra-abdominal and pelvic sepsis following gastrointestinal surgery."

Tracy, O., et al. (29 Jan.'72).
Brit. med. J., p. 280.

"Our experience with this series of seriously ill patients provides clinical confirmation to complement recent in vitro evidence that clindamycin is the antibiotic of choice for use in bacteroides infections. Not only was the response in 17 of the 18 patients favourable, but in several it was dramatic."

Haldane, E.V. and van Rooyen, C.E. (1972).
C.M.A.J., p. 1177.

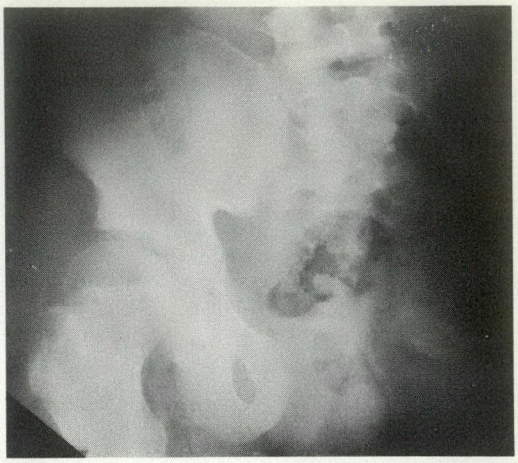


Fig. 6.—Appearance of right hip and amputated left hip region 7 years after hemipelvectomy.

The present case, however, does appear to be an example of malignant transformation of a chondromyxoid fibroma. Chondrosarcomatous degeneration was documented in a proven chondromyxoid fibroma of bone. Fig. 5 shows that the abrupt fibrous boundaries of chondromyxoid fibroma are lacking and the the tumour is frankly malignant—in contrast to the benign radiographic and histologic appearance of the original tumour (Figs. 1 and 2).

We thank Dr. J. M. McIntyre, who allowed us to review this patient, and the department of pathology and the audiovisual department.

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PROPHYLACTIC ROLE OF CEPHALORIDINE IN SURGICAL WOUNDS

P. K. SURI, MB, BS* and D. W. B. JOHNSTON, MD, FRCS[C], FACS†

Summary: The incidence of postoperative wound infection was studied in relation to 146 surgical wounds in patients who received cephaloridine as prophylactic antibacterial therapy, and in relation to 120 wounds in another group of patients who did not receive cephaloridine. In the first group, 5 of 146 (3.4%) of wounds became infected; in the second, 25 of 120 (20.8%) of wounds became infected. The difference between the two groups is significant. Use of cephaloridine in this series of patients therefore decreased the incidence of postoperative wound infection.

Résumé: Nous avons étudié la fréquence des infections de plaies opératoires sur 146 plaies opératoires pratiquées sur des patients qui avaient reçu de la céphaloridine comme traitement antibactérien préventif et, également, sur un autre groupe de 120 patients qui, eux, n'avaient pas reçu de céphaloridine. Nos constatations: dans le premier groupe, infections des plaies dans 5 cas sur 146, soit dans 3.4% des cas. Dans le second groupe, 25 des 120 plaies opératoires (soit 20.8%) devinrent infectées. La différence entre les deux groupes est considérable. Il s'ensuit que, dans cette série de patients, le recours à la céphaloridine a diminué la fréquence de l'infection post-opératoire des plaies chirurgicales.

USE of antimicrobial agents in the prophylaxis of infection implies that the microorganisms are attacked either during the period of contamination before colonization has occurred, or, if colonization has taken place, before invasive infection begins. The prophylactic use of modern antibacterial drugs has been put to the test by controlled prospective clinical observations, by retrospective analysis, and by animal experimentation. Yet, not all surgeons agree on their effectiveness. Findings both for[1] and against[2, 3] prophylactic antibiotic therapy have been reported. Among authors in favour are Polk and Lopez-Mayor,[1] who found that cephaloridine reduced the incidence of postoperative wound sepsis.

Drug prophylaxis has no place in operative procedures that carry minimal risk relative to sepsis. Conversely, drug prophylaxis may reduce the incidence and severity of infection in certain conditions in which risks are great. Examples of these latter conditions are the following:

1. Wound resulting from trauma.
2. Burns.
3. Operative sites associated with heavy contamination.
4. Preparation of large intestine for operations.
5. Any operation requiring the insertion of a permanent prosthesis.
6. Surgical procedures on patients prone to infection because of factors such as impoverished local blood supply, the carrier state, undernutrition, preexisting infection remote from the operation site or therapy that may alter host defence mechanisms (i.e. immunosuppressive agents).

Davidson, Clark and Smith[4] made a valuable contribution by classifying wound infections into three major divisions. Altemeier, Culbertson and Hummell[3] found that bacterial contamination of wounds during operation and infrequently ward conditions were important factors. Other predisposing causes such as obesity[5] and long preoperative stay in hospital[2] also contributed to wound sepsis.

In this paper we report the results of a study undertaken to clarify the role of antibiotics, specifically cephaloridine, in the prevention of postoperative wound sepsis. Cephaloridine was chosen as our specific antibiotic because of its unique characteristics as an inhibitor of gram-negative and gram-positive organisms, the relatively few contraindications, the absence of pain with intramuscular injection, and its relatively low toxicity.[6]

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METHODS

A group of 146 surgical wounds was studied. The "coin test" was used to determine which patients should receive cephaloridine. A coin was tossed for each patient. A patient would receive cephaloridine whenever the coin fell with the head side uppermost (a positive coin test[1]). Three injections of 1 g each were given, the same doses as given by Polk and Lopez-Mayor[1] in positive coin test. The first dose was given intravenously during induction of anesthesia. The second dose was administered intramuscularly 5 hours after the operation and the third dose intramuscularly after 12 hours. If the operation entailed two incisions these were counted separately; this accounts for the discrepancy between the number of wounds and the number of patients. A control group comprised 120 wounds. The distribution of surgical procedures is presented in Table I.

Wounds were inspected 24 hours after operation and daily thereafter until healing was complete. Impartial observers were asked to carry out these observations and no single observer was given the total responsibility. If there was any evidence of wound infection in the form of inflammation, discharge, or edema of edges, a swab was sent for culture and sensitivity. Wound infection was no contraindication to administration of another antibiotic.

The wounds were divided into two categories: clean and contaminated. Contamination is more likely to occur in large bowel operations than cholecystectomy or orthopedic procedures. Wound contamination was further related to a low expectation of infection and a high expectation of infection, categories that Jackson, Pollock and Tindal[7, 8] and also Pollock and Tindal[9] termed potentially contaminated and contaminated.

The results were analyzed by means of the chi-square test.

RESULTS

Of the 146 wounds in patients who received cephaloridine, 5 (3.4%) became infected. Of the control group of 120 wounds in patients who were not given cephaloridine, 25 (20.8%) became infected (Table II). In this series, therefore, use of cephaloridine significantly decreased the incidence of postoperative wounds.

TABLE II.—ANALYSIS OF RESULTS

Treatment group	*Infection**	*No infection**	*Total*
Treatment	5 (3.4%)	141	146
No treatment	25 (20.8%)	95	120
Total	30	236	266

*Difference statistically significant by chi-square analysis ($P = < 0.001$, $df = 1$).

TABLE I.—DISTRIBUTION OF OPERATIONS IN RELATION TO CLEANLINESS OR CONTAMINATION OF WOUND AND TO TREATMENT WITH CEPHALORIDINE

	Distribution of operations with respect to cephaloridine treatment			
	Treated		*Not treated*	
Nature of operation	*No.*	*%*	*No.*	*%*
Contaminated wounds	46	31.5	40	33.3
Colorectal procedures	40	27.4	36	30.0
Other procedures	6	4.1	4	3.3
Clean wounds				
Groin herniorrhaphy	22	15.1	13	10.8
Varicose vein procedures	30	20.6	27	22.5
Amputation	19	13.0	16	13.3
Lumbar incision procedures	4	2.7	3	2.5
Nonarterial procedures	16	11.0	11	9.2
Arterial procedures	9	6.2	10	8.3
Total	146*	100.0	120	100.0

*Some operations entailed more than one wound.

Discussion

We have shown that treatment with cephaloridine is a significant factor in the prevention of wound infection in all types of wounds; that is, in clean wounds other than those for arterial disease of the lower extremities, in grossly contaminated wounds, and in contaminated wounds other than those for colorectal surgery.

Our study tends to confirm the work of Polk and Lopez-Mayor.[1] Evans and Pollock[10] likewise reported on lower incidence of wound infection after using cephaloridine.

Polk and Lopez-Mayor[1] studied both wound and intra-abdominal infections. They gave cephaloridine prophylactically to 32 patients with gastroduodenal operations and none of the wounds in these patients became infected, whereas in 36 patients given a placebo, 11 infections occurred. Cephaloridine was also used in association with 54 colorectal operations; 4 wound infections were reported, in contrast to 15 wound infections in 50 patients undergoing colorectal operations who did not receive cephaloridine.

Cephaloridine is a bactericidal antibiotic active against *Staphylococcus pyogenes* and other gram-positive cocci (only some strains of *Streptococcus fecalis).* The gram-negative organisms that are sensitive to cephaloridine include *Proteus mirabilis, Escherichia coli* and most strains of *Klebsiella pneumoniae.* It is ineffective against *Pseudomonas aeruginosa.*

In the present series, administration of cephaloridine did not result in change of organism or in the emergence of cephaloridine-resistant organisms.

We gratefully acknowledge the statistical advice offered by Dr. Charles Rand, University of Western Ontario, London, Ont.

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INDICATIONS

The control of pathogenic organisms which may infect tissues exposed during surgery, and contaminate wounds and burns. Useful in casualty and pelvic surgery, open orthopedic and neurological procedures, as well as in general abdominal, pelvic and thoracic surgery, and the treatment of burns.

PRECAUTIONS

Prolonged use of antibiotics may result in the overgrowth of non-susceptible organisms, including fungi. Appropriate measures should be taken if this occurs.

DOSAGE

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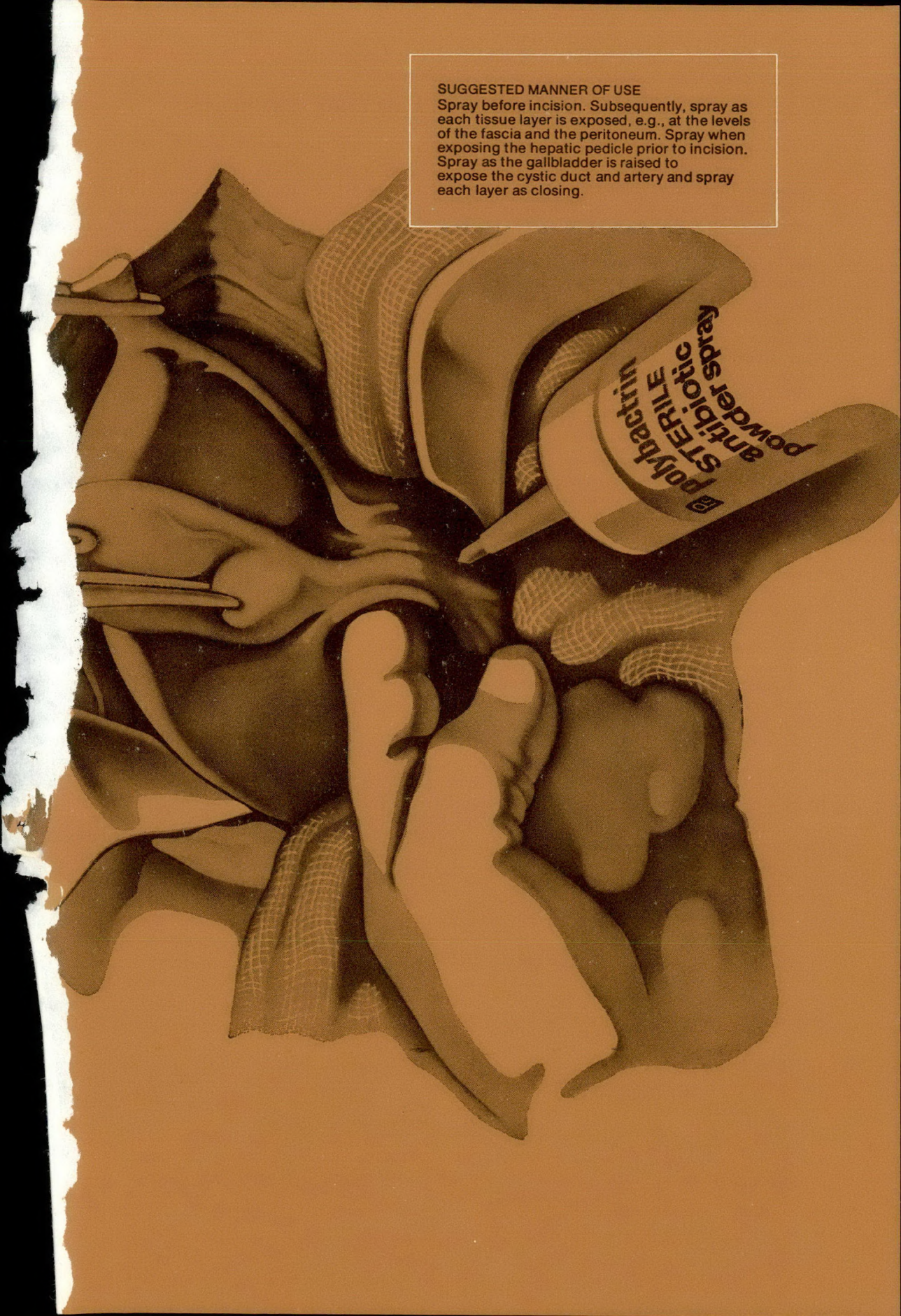

COMPLICATIONS OF CLOSURE OF LOOP COLOSTOMY*

W. W. YAKIMETS, MD, FRCS[C], FACS†

Summary: Among 71 patients undergoing loop colostomy closure, one complication or more developed after operation in 35. Most of the complications were not serious, but there were two anastomotic leaks and two patients died. Some of the complications may be prevented by avoiding the use of drains and by packing the skin and subcutaneous tissues open with Vaseline gauze. There does not appear to be any advantage in using antibiotics in preoperative bowel preparation. A one-layer intraperitoneal colostomy closure can be done safely but is frequently associated with minor complications.

Résumé: Sur les 71 malades chez lesquels on avait pratiqué la fermeture d'une anse de colostomie, au moins une complication post-opératoire est survenue chez 35 opérés. La majorité de ces complications n'étaient pas graves, bien qu'on ait constaté deux cas de fuites au niveau des anastomoses et le décès de deux malades. Certaines complications peuvent être empêchées en évitant les drains et en utilisant des tampons de gaze vaselinée dans la plaie opératoire. Le recours préopératoire aux antibiotiques pour préparer l'intestin ne semble présenter aucun avantage. La fermeture intrapéritonéale d'une colostomie en une couche peut être faite sans danger, mais elle donne souvent lieu à des complications mineures.

Loop colostomy closure is a common elective surgical procedure. A study of the complications of this operation may help surgeons to understand and to reduce the complications of colonic surgery in general.

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Reports of complications of closure of loop colostomy by different authors are summarized in Table I.[1-4] Because several of these authors, especially Knox, Birkett and Collins,[2] have reported many serious complications after colostomy closure, all loop colostomy closures carried out at the University of Alberta Hospital in the 5-year period from January 1968 through December 1972 were reviewed.

Patients and Surgical Details

During the period January 1968 through December 1972, 71 patients (38 male and 33 female) underwent loop colostomy. The primary disease was carcinoma of the colon in 21 patients, diverticular disease in 25 patients and miscellaneous diseases in the remaining 25 (Table II). In 63 patients colostomy was performed either before a definitive procedure to relieve obstruction or during a definitive procedure to protect the anastomosis; in the remaining 8 patients the colostomy was done after resection of the colon when leaks occurred at the anastomosis. The colostomy was of the right transverse colon in 60 patients, of the left transverse colon in 7, of the middle transverse colon in 1, and of the sigmoid colon in 3. Before closure of the colostomy in 27 of the 46 patients who had previously undergone colon resection for carcinoma or diverticular disease, the anastomosis was checked by a barium enema or sigmoidoscopy, or both. A localized leak at the anastomosis was demonstrated in five patients, and this delayed closure of the colostomy. Healing of the anastomoses in the miscellaneous group was not assessed in the study due to a variety of non-comparable

TABLE I.—Reported Complications of Colostomy Closure

Author	*Total no. of patients*	*No. of complications*	*No. of infections*	*No. of leaks*	*No. of deaths*
Barron and Fallis[1]	200	?	Few	1	0
Knox, Birkett and Collins[2]	179	115	18	42	4
Thomson and Hawley[3]	191	150	32	7	0
Bell[4]	284	?	?	18	5

TABLE II.—Indications for Loop Colostomy in 71 Patients

Indication		*No. of patients*
Carcinoma		21
Diverticular disease		25
Miscellaneous		25
Ischemic colitis	6	
Penetrating injuries	5	
Imperforate anus	5	
Fistulas	4	
Anastomotic leaks	3	
Villous adenoma	2	
Total	25	

TABLE IV.—Methods of Loop Colostomy Closure in 71 Pateints

Details of anastomosis	*No. of patients*
Edges	
Excised	69
Intact	2
Closure	
Intraperitoneal	70
Extraperitoneal	1
Anastomosis	
One layer	70
Two layers	1
Suture	
Silk	63
Steel	4
Catgut	1
Unspecified	3

definitive procedures—for example, imperforate anus.

Preparation of the bowel before closure of the colostomy varied with the surgeon (Table III). The methods were of four categories. All patients received a diet of clear fluids and mechanical cleansing for 2 or 5 days; in addition, some patients received antibiotics or sulfonamides, or both. All but one of the closures were intraperitoneal. The anastomotic technique was similar in most, but not all, of the procedures, a single layer of simple 3-0 silk sutures being used after the edges of the colostomy had been excised (Table IV).

Wound infections (any wound discharging pus) were studied in relation to the type of preoperative bowel preparation, wound drainage and wound closure. The shortest follow-up period was 12 months.

The "*z*" test was used in the statistical analysis of the results.[5]

Results

Complications

One complication or more developed after loop colostomy closure in 35 patients (49%) (Table V); wound infection, prolonged ileus and incisional hernia were the commonest. In the two patients with anastomotic leaks wound infection and wound dehiscence also developed. One of these patients required a further procedure to close the fistula; in the other, the fistula closed spontaneously. Neither of these two patients died.

Among the total of 71 patients, 2 died, but in only 1 could death be attributed directly to the operation. In one woman severe diarrhea developed 11 days after colostomy closure, and this was followed by rectal bleeding; this patient died of septic and hypovolemic shock 22 days after colostomy closure, necrosis of the colonic mucosa being discovered at autopsy. Another woman died from a cerebrovascular accident 7 days after operation.

There was no significant difference in the rate of complications between males and females and between the various age groups.

Effect of bowel preparation.—The addition of antibiotics to mechanical methods of bowel preparation did not significantly diminish the incidence of wound infection

TABLE III.—Bowel Preparation and Complications

Duration of preparation (d)	*Regimen*	*No. of patients*	*No. of infections*
5	Mechanical	30	13
	Mechanical and antibiotic	12	3
2	Mechanical	27	10
	Mechanical and antibiotic	2	1
	Total	71	27

TABLE V.—COMPLICATIONS

Complication	*Diagnosis*			*No. of complications**
	Carcinoma	*Diverticulitis*	*Miscellaneous*	
Wound infection	5	9	13	27
Incisional hernia	3	4	3	10
Ileus	2	0	5	7
Dehiscence	1	0	1	2
Anastomotic leak	1	0	1	2
Rectal hemorrhage	0	1	0	1
Fecal impaction	0	0	1	1
Total	12	14	24	50

*Some patients had more than one complication.

(P = 0.13). Nor was there any evidence that prolonging the mechanical bowel preparation for more than 2 days decreased the infection rate (P = 0.32).

Effect of suture techniques.—Because most of the colostomies were closed in a similar fashion, one cannot compare the method of closure in these patients with any other method. However, because there were only two instances of leakage at the suture line (2.8%), the method used in these patients appears to have been satisfactory. By comparison, Thomson and Hawley[3] reported a suture-line leak in 2.9% of patients in whom a double layer of catgut and silk was used in the intraperitoneal anastomosis without the edge of the colostomy being excised in the majority of cases. This implies that a carefully constructed anastomosis is reasonably safe, irrespective of the method.

Effect of wound drainage.—All six operations in which an intraperitoneal drain was used were followed by complications. Two of these complications were leaks at the suture line (Table VI). This high complication rate is probably attributable to the use of an intraperitoneal drain in an attempt to prevent more serious complications in patients with a doubtful anastomosis or serious contamination. Statistically there was no evidence that a subcutaneous drain reduced wound infections.

Effect of method of wound closure.—There was no significant difference in the infection rate of wounds that were packed open compared to those that were sutured (P = 0.09) (Table VII). Leaving the skin and subcutaneous tissues open does not appear to weaken the wound, because an incisional hernia developed in only one of the unsutured wounds. Although the numbers are small, the incidence of incisional hernias is less in open infected compared with closed infected wounds (P = 0.05).

Duration of hospital stay.—Complications increased the average stay from 10.4 to 19.6 days (Table VIII).

TABLE VI.—DRAINS AND COMPLICATIONS

Wound drainage	*No. of Patients*	*No. of infections*	*No. of hernias*	*No. of leaks*
Intraperitoneal	6	6	0	2
Subcutaneous	17	7	3	0
None	48	14	7	0
Total	71	27	10	2

TABLE VII.—SKIN APPROXIMATION AND COMPLICATIONS

Skin approximation in wound	*No. of Patients*	*No. of infections*	*No. of hernias*
Sutured	43	19	9
Open	28	8	1
Total	71	27	10

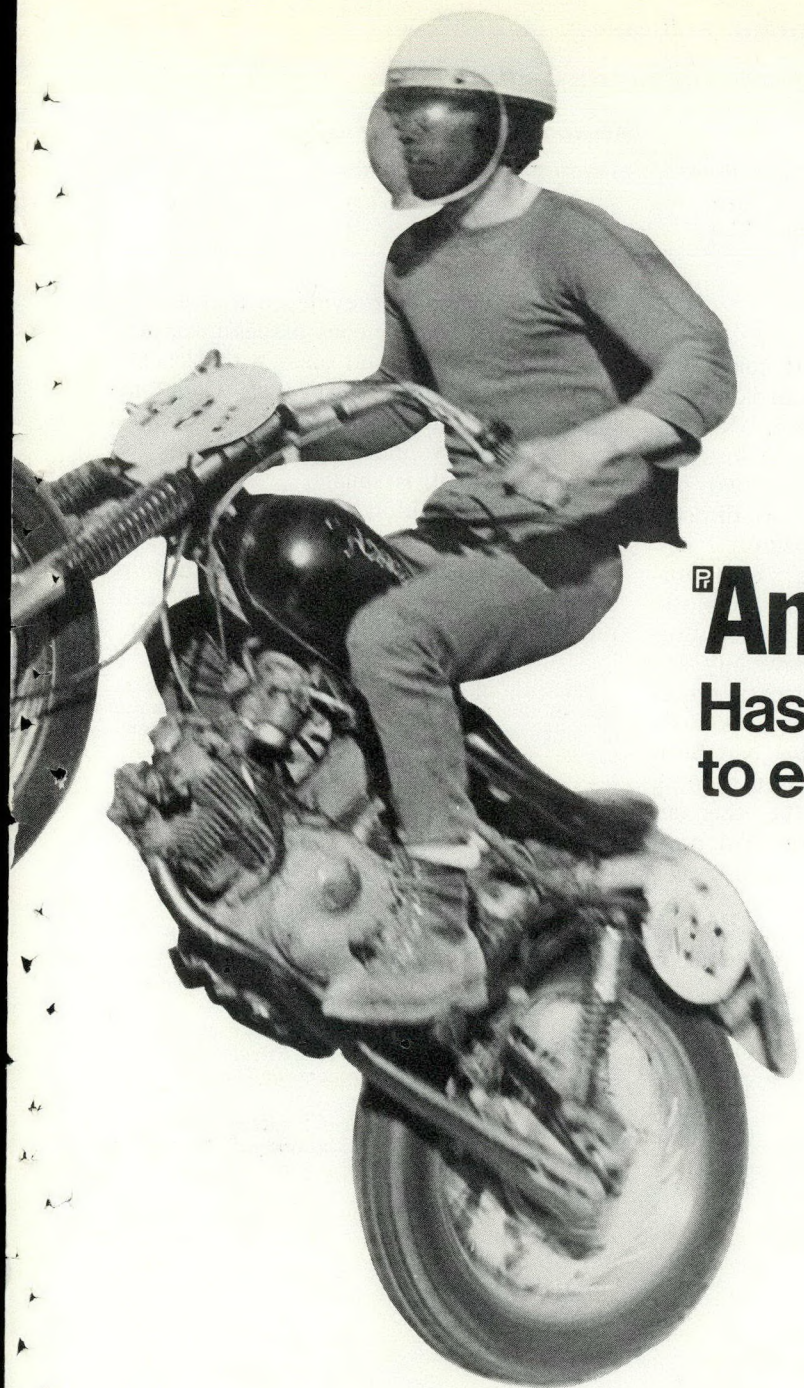

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TABLE VIII.—AVERAGE DURATION OF HOSPITAL STAY

Postoperative course	*Average duration of hospital stay (d)*			
	Carcinoma	*Diverticulitis*	*Others*	*Total*
Uncomplicated	10.8	10.3	9.9	10.4
Complicated	21.5	13.1	24.9	19.6

DISCUSSION

This investigation shows that colostomy closure is not innocuous and that it carries a high complication rate (49%). Most of the complications were not serious but two were suture-line leaks. Moreover two patients died, and death in one was directly attributed to the colostomy closure.

Certain of the findings are worth emphasizing. First, the use of an antibiotic plus mechanical preoperative bowel preparation does not appear to have any advantage over mechanical bowel preparation alone: one of the anastomotic leaks occurred in a patient whose bowel had been prepared for 5 days by antibiotic therapy and by mechanical means and the other occurred in a patient whose bowel had been prepared over 2 days by mechanical means only.

Second, intraperitoneal bowel closure seems to be safe, and there is no evidence that the technique of colon anastomosis or the suture material used has any bearing on the development of complications.

Third, intraperitoneal drains are associated with a high complication rate, probably because of their use in patients in whom complications are expected.

Fourth, there is no evidence that leaving the skin and subcutaneous tissues unsutured and packing the wound open with Vaseline gauze reduces the incidence of wound infections. The incidence of incisional hernia in unsutured wounds, even in those that become infected, is small.

Finally, in addition to the increased morbidity, complications are costly. The hospital stay was nearly doubled in patients with complications and 10 patients were readmitted later for repair of incisional hernia.

The help of Dr. Betty Williams in the preparation of this paper and of Mr. Wayne Osbaldeston in preparing the statistics is gratefully acknowledged.

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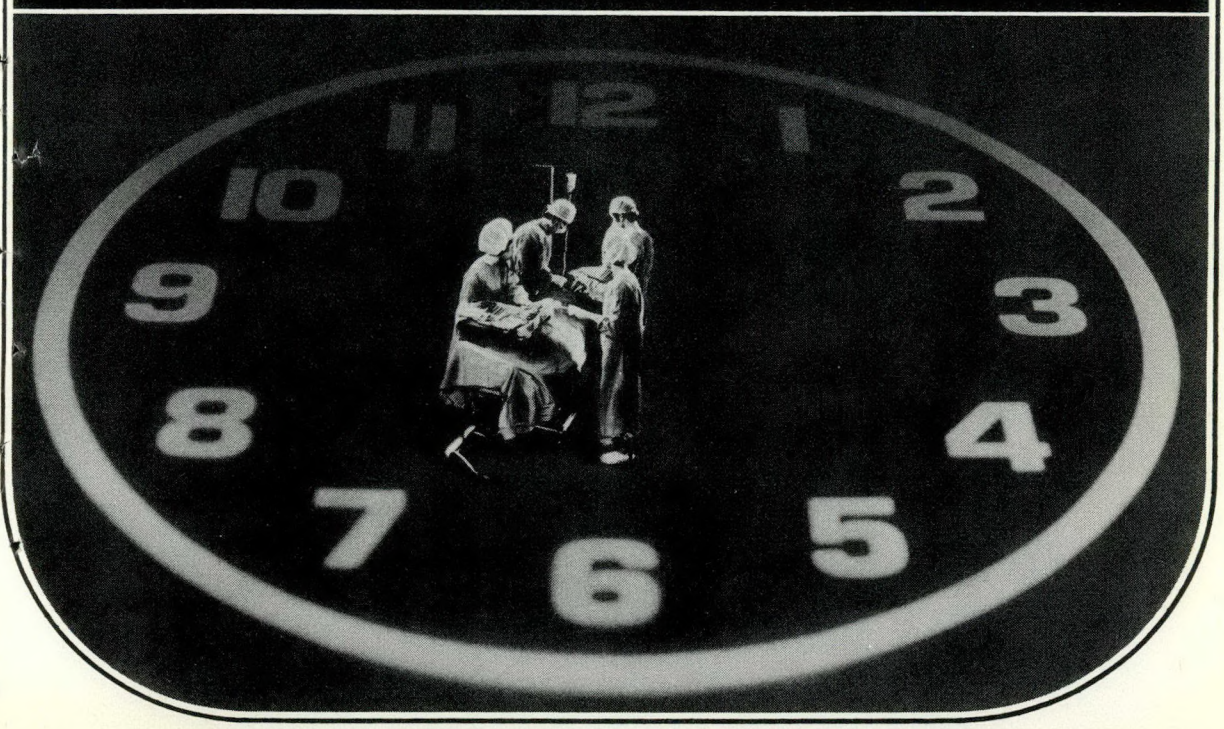

BACTERIAL PERITONITIS AND THE BURSTING STRENGTH OF INTESTINAL ANASTOMOSES

S. K. KILAM, MB, FRCS[C],* F. L. JACKSON, MD, DipBact(Lond), MCPath† and H. T. G. WILLIAMS, MB, FRCS[C], FACS‡

Summary: In order to study the bursting strength of intestinal anastomoses, resection of a wedge of ileum and creation of an anastomosis were performed, and peritonitis was induced in five groups of rats. Peritonitis was induced by intraperitoneal injection of bacterial suspensions of *Escherichia coli, Pseudomonas aeruginosa, Staphylococcus aureus* and *Clostridium welchii*, and the exteriorized ileal loop in each rat was regularly inspected for peritoneal exudation, adhesions, hyperemia, obstruction, leakage and bursting strength. All bowel anastomoses performed in the presence of peritonitis were weaker than those done in a sterile field (control group) and the type of organism influenced the strength of the anastomosis and the histopathologic changes; in this study the weakest anastomoses were associated with *E. coli* infection.

Résumé: En vue d'étudier la résistance à l'éclatement d'anastomoses intestinales, nous avons procédé, chez cinq groupes de rats à la résection cunéiforme de l'iléum et à la création d'une anastomose. Puis nous avont provoqué une péritonite artificielle par injection intrapéritonéale de suspensions bactériennes des organismes suivants: *Escherichia coli, Pseudomonas aeruginosa, Staphylococcus aureus* et *Clostridium welchii*. Nous avons alors inspecté, chez tous les animaux, l'anse iléale extériorisée, notamment l'exsudation péritonéale, les adhérences, l'hyperhémie, l'occlusion, les fuites et la résistance à l'éclatement. Toutes les anastomoses intestinales pratiquées en présence de péritonite étaient plus faibles que celles qui avaient été faites dans un champ stérile (groupe témoin). La nature du micro-organisme influençait la résistance de l'anastomose et les modifications histopathologiques dans notre étude, les anastomoses les plus faibles étaient celles qui avaient été infectées par *E. coli*.

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INFECTION is a major cause of impaired healing of surgical wounds, and the degree of impairment varies with the type of infecting organisms, size of inoculum and various local and systemic factors.

Letwin and Williams[1] showed that peritonitis diminished the bursting strength of the anastomosis by 50 to 75% compared with a normally healing anastomosis tested on the 5th postoperative day. Although the relation between the inflammatory response to local trauma and subsequent wound healing has long been recognized, little information is available regarding the effect of specific bacteria on healing of intestinal anastomosis. Smith and Enquist[2] studied the effects of standardized staphylococcal infections on sutured musculofascial incisions in rats and compared the results with uninfected controls. They found that infected wounds contained less collagen and more hexosamine and that they were weaker than the wounds of controls. Meleney et al[3] found that pathogenicity was increased in cases of mixed infection. Condie and Ferguson[4] observed that a decrease in dead space in wounds reduced the chances of infection.

We report the results of a study of the effect of peritonitis induced by standardized bacterial suspensions on the bursting strength of intestinal anastomosis in rats.

METHOD

Adult, male, Sprague-Dawley rats (weight range, 230 to 250 g) were used. (The use of rats is economical and numbers of genetically and physiologically similar animals of the same strain could be provided.) Animals were caged individually for at least 4 days before operation and kept on a standard diet. No food was given for 24 hours before operation but water *ad libitum* was allowed. The animals were anesthetized by intraperitoneal injection of pentobarbital sodium (3 to 4 mg/100 g). The operative procedure was as follows: Through a midline incision (length, 4 cm), under aseptic conditions, a loop of ileum was extruded and, after liga-

tion of one of the small arcades of mesenteric vessels, wedge resection of the relatively ischemic segment was performed. An end-to-end, single-layer anastomosis was performed with interrupted 6-0 silk sutures. Usually, three or four sutures were used on each side, depending on the size of the lumen of the bowel. Contamination of the peritoneal cavity by the intestinal contents and blood clots was avoided as much as possible. The abdomen was closed in two layers with 3-0 chromic catgut. For the first 12 hours after operation, water was allowed *ad libitum* but no food was given.

For the study of the effect of peritonitis, the rats were divided into five groups of 18. In the control group, the abdomen was closed without introduction of any bacterial suspension. In each of the other groups, 2 ml of a bacterial suspension, known by preliminary trial to produce active peritonitis without being lethal, was introduced into the peritoneal cavity after the anastomosis had been completed and before the abdomen had been closed. Bacteriologic methods were as follows: Bacteria were grown for 18 hours in trypticase-soy broth, collected by centrifugation, suspended in Ringer's solution and stored at 4°C. Viable counts were stable over a period of 4 days, as determined by the method of Miles, Misra and Irwin.[5] Bacterial suspensions were then adjusted to contain the necessary number of viable organisms (based on preliminary tests). The organisms used were *Escherichia coli, Pseudomonas aeruginosa, Staphylococcus aureus* and *Clostridium welchii;* the concentrations of these organisms in suspension were, respectively, 10^7/ml, 10^6/ml, 10^6/ml and 10^8/ml.

The anastomoses were inspected daily after the operation up to 11 days. Then, after intraperitoneal injection of a lethal dose of pentobarbital sodium, the abdomen was immediately opened and studies of gross lesions, the bursting strength of the anastomoses, and microscopic lesions at the anastomosis were made.

Gross Intra-abdominal Lesions

Lesions sought included peritoneal exudation, adhesions, hyperemia, and obstruction and leakage.

Bursting Strength of the Anastomosis

The bursting pressure of the anastomosis in our experiments was studied after the anastomotic site together with 3 cm of normal intestine on each side had been removed. Adhesions were not removed completely and excessive handling of the tissues was avoided. One end of the bowel was connected to a tube with a manometer and a reservoir of coloured water in parallel. Coloured water was instilled gently into the segment of bowel, the source of pressure being a steady stream of air introduced into the water reservoir at a constant rate and pressure. The whole preparation was kept in a tray of clear water and, after removal of air bubbles, the open end of the bowel was clamped with a hemostat. The first evidence of leakage of coloured water from the bowel segment at or around the site of anastomosis was recorded as the bursting pressure strength (in mm Hg).

Histologic Studies of the Anastomotic Sites

Studies included counts of different types of cells in areas of 20 mm^2, and estimations of the amount of epithelial regeneration and of the amount of ground substance. Routine histologic stains were used in tissue staining. Both sides of the anastomotic site were examined.

Results

Gross Intra-abdominal Lesions

In the control group of animals, few adhesions and few signs of peritoneal exudation were noted. There was slight evidence of edema and ulceration and no signs of obstruction or leakage at the anastomotic site.

In the rats in which peritonitis had been induced with different bacteria, the gross changes in the abdominal cavity were those summarized in Table I.

Bursting Strength of Anastomoses

The degree of weakness in the bursting strength of the anastomosis was related to the presence of infection (Table II) and was dependent on the type of organism causing the infection (Figs. 1 and 2). The

TABLE I.—Gross Lesions Induced by Intraperitoneal Injection of Bacteria

Lesion	Organism			
	E. coli	*Ps. aeruginosa*	*S. aureus*	*Cl. welchii*
Peritoneal exudate	Moderate: hemorrhagic	Marked: hemorrhagic	Moderate: serous	Marked: hemorrhagic
Adhesions	Few initially: moderate after 7th day	Moderate	Marked	Few
Hyperemia	Moderate	Slight	Slight	Marked
Obstruction or leakage	Moderate obstruction: no leakage	None	None	Marked

TABLE II.—Bursting Pressures (mm Hg) of Anastomoses in Relation to Absence or Presence of Intraperitoneal Infection and to Postoperative Day

Postoperative day	Bursting pressures (mm Hg) for five experimental groups				
	Control	*E. coli*	*Ps. aeruginosa*	*S. aureus*	*Cl. welchii*
1	70*	30	30	30	30
	100*	40	40	60	60
	120*	60	40	80	75
3	160	60	40	80	100
	160	80	80	80	140
	180	80	80	90	140
5	165	100	70	90	160
	170	120	100	210	170
	200	140	120	260	180
7	220	180	175	230	200
	260	200	180	240	250
	275	200	250	250	260
9	290	240	200	240	200
	295	260	210	250	260
	300	260	280	270	280
11	290	260	170	200	240
	300	260	260	260	260
	300	270	280	280	280

*Entries in each line relate to findings in one rat.

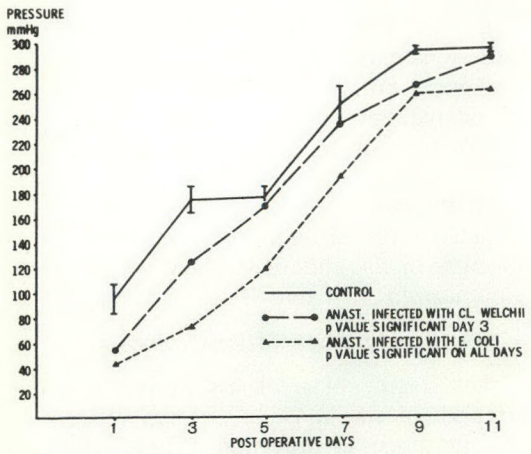

Fig. 1.—Relationship of bursting pressure of intestinal anastomosis with postoperative days in rats infected with *S. aureus* and *Ps. aeruginosa*.

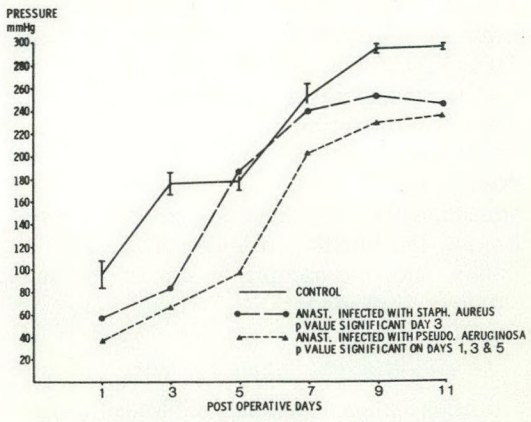

Fig. 2.—Relationship of bursting pressure of intestinal anastomosis with postoperative days in rats infected with *Cl. welchii* and *E. coli*.

anastomosis was weakest in rats in which infection had been induced by intraperitoneal injection of *E. coli* and *Ps. aeruginosa*. The anastomoses did not attain the same bursting strength as the anastomoses in the control group of rats up to the 11th day.

Histologic Changes

Up to the 4th postoperative day, an increase in wound strength coincided with an increase in the number of fibroblasts. The numbers of macrophages and round cells decreased after the 4th day.

There was an impressive difference in the numbers of polymorphonuclear cells between normal and infected anastomoses. In normal anastomoses the initial polymorphonuclear cell count was very high; the number of polymorphonuclear cells decreased sharply over the first 3 days and coincided with a rapid increase in strength of the wound. This did not occur in any of the rats in whom the wounds healed with surrounding peritonitis; in these animals the polymorphonuclear cell count remained low.

Discussion

Distension of a healing intestinal anastomosis as a measure of its strength was first used by Chulmusky.[6] Harvey and Howes[7] used this method to study the influence of diet on the healing of stomach wounds in rats. Herrmann, Woodward and Pulaski[8] made elaborate studies on the healing of colonic anastomosis in rats and used bursting strength as a measure of healing; they concluded that the biophysical and morphologic parameters are generally correlative and complementary. We found that the bursting strength of anastomoses of rats with peritoneal infection was lower from the beginning and remained low up to the 11th postoperative day. The critical period of healing in an anastomosis seems to antedate the 5th day; it is therefore important to take complete precautions during this period, especially when the bowel anastomosis is performed in the presence of peritonitis: nothing should be given by mouth and the bowel should be kept empty and at rest.

The gross lesions noted in the peritoneal cavity varied with the type of organism causing peritonitis. Only slight hyperemia of the bowel was noted in the presence of peritonitis induced by *Ps. æruginosa* and *S. aureus*. There was no evidence of obstruction or leakage at the anastomotic site in rats with this type of infection.

Moderate to marked hyperemia of the bowel, with minimal adhesion formation, was seen in rats with peritonitis induced by injection of *E. coli* and *Cl. welchii*. There was a moderate amount of hemorrhagic peritoneal exudate and some degree of bowel obstruction in these animals. This relation between marked hyperemia of the bowel, moderate hemorrhagic peritoneal exudate and bowel obstruction probably can be attributed to persistence of an acute inflammatory process, which could also lead to poor adhesion formation around the anastomosis, especially in the presence of infection.[9]

No explanation can be offered for the small degree of hyperemia and marked hemorrhagic peritoneal exudate in rats infected with *Ps. aeruginosa*.

A clinical implication of these findings is that identification of the causative organism may be helpful in assessing prognosis, and also in determining the treatment of patients in whom bowel anastomoses are performed in the presence of infection.

The role of blood cellular elements, especially polymorphonuclear cells, in the healing process is a source of speculation. The high initial counts of polymorphonuclear cells with rapid decrease after the 3rd day compared with the relatively low polymorphonuclear count in animals with infected wounds cannot be explained. This depression was most apparent in rats with *E. coli* infection. As well as having phagocytic activity, the polymorphonuclear cells probably have some effect on fibroplasia and thus on the strength of wound.

The number of animals examined was too small for valid statistical analysis; however, the results warrant further investigations.

Conclusions

Based on bursting pressure studies of in-

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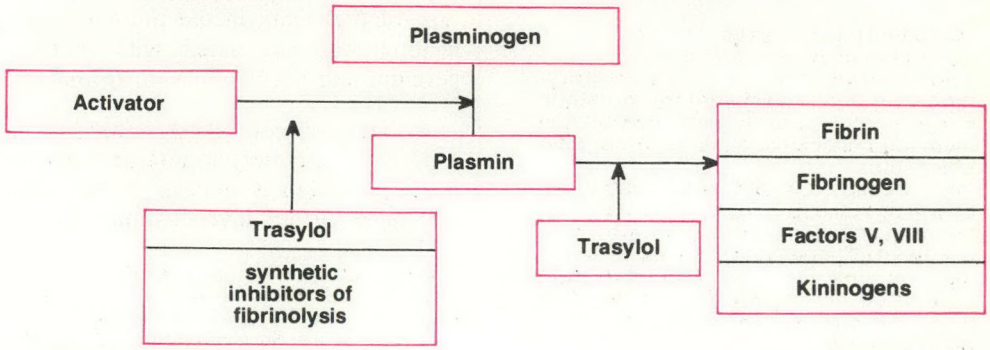
Trasylol® ..... 7.5% Mortality

"Because the number of deaths were reduced, the spectrum of the disease as a whole was altered." *

Trapnell's recent double-blind clinical trial involving 105 patients confirmed the effectiveness of Trasylol in acute pancreatitis. In addition to reducing mortality and altering the spectrum of the disease, Trasylol largely abolished the usual effect of increasing age in this condition. Trasylol should be given concurrently with the usual measures for the treatment of pancreatitis, such as pain relief, fasting, gastric suction, etc. "It (Trasylol) can therefore now be regarded as a drug which is both effective and beneficial in the treatment of acute pancreatitis." *

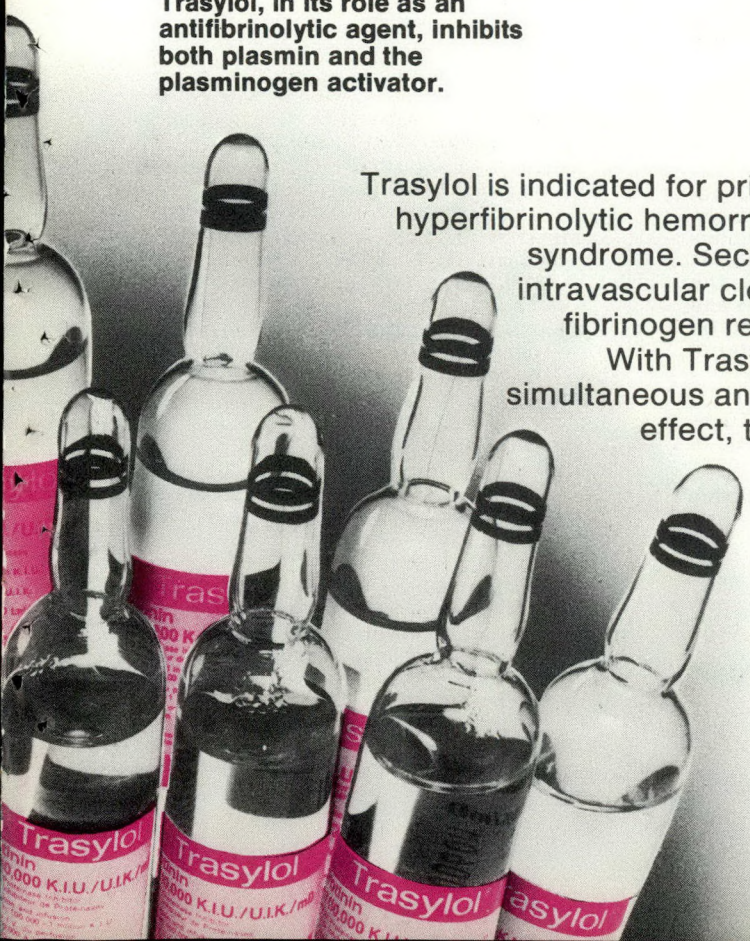
*Trapnell, J.E. et al, British J. Surg., March 1974.

The Effect of Trasylol® in Hyperfibrinolytic Hemorrhages



Trasylol, in its role as an antifibrinolytic agent, inhibits both plasmin and the plasminogen activator.

Synthetic antifibrinolytic agents inhibit only the plasminogen activator.



Trasylol is indicated for primary and generalized, secondary hyperfibrinolytic hemorrhages with and without the shock syndrome. Secondary hyperfibrinolysis following intravascular clotting may require heparin and/or fibrinogen replacement in addition to Trasylol. With Trasylol, the antifibrinolytic agent with simultaneous anti-coagulant (anti-thromboplastic) effect, there will not be a dangerous shift in the equilibrium between the clotting and the lysis system in favour of coagulation. Synthetic antifibrinolytics are contraindicated if there is evidence of active intravascular coagulation. Trasylol is indicated in all cases of hyperfibrinolysis, whether a differential diagnosis has been made or not.

FBA Pharmaceuticals Ltd.
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testinal anastomoses in rats, the following conclusions can be drawn:

1. All bowel anastomoses performed in the presence of peritonitis are weaker than those done in a sterile field (control group).

2. The type of organism causing the peritonitis directly affects the strength of the anastomosis, in the present study the weakest anastomosis was associated with *E. coli* infection.

3. Gross pathologic changes found in the peritoneal cavity appear to vary with the type of organism; in the present study poor healing was associated with marked hyperemia and scant adhesion formation.

4. The role of blood cellular elements, especially polymorphonuclear cells, in the healing process is unclear at this time and will need further investigation.

We thank the Edmonton Civic Employees' Association and the University of Alberta Medical Research Fund for financial assistance. The work was done in the Surgical-Medical Research Laboratory, University of Alberta.

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7. Harvey SC, Howes EL: Effect of high protein diet on velocity of growth of fibroblasts in healing wound. *Ann Surg* 91: 641, 1930
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CORRESPONDENCE

CYSTIC ADVENTITIAL DISEASE OF POPLITEAL ARTERY

To the Coeditors:

In their interesting paper entitled "Cystic Adventitial Disease of the Popliteal Artery" *(Can J Surg* 18: 46, 1975), Drs. T. K. Scobie and R. H. Curry reported what they believed to be "The first case of cystic adventitial disease of the popliteal artery recorded in Canada". As it happens, I reported a case in 1969 (LAURENDEAU F: La dégénérescence adventicielle kystique de l'artère poplitée. *Union Med Can* 98: 589, 1969), so that the assumption of Drs. Scobie and Curry that theirs is the first Canadian report is incorrect.

I was, however, glad to see another report of this rare condition, because to assert that one is reporting the first case of any disease, even if the claim is limited to the Canadian literature, is always hazardous.

F. LAURENDEAU, MD, FRCS[C]

Chief, department of surgery,
Maisonneuve-Rosemont Hospital,
Montréal, Qué.

To the Coeditors:

We appreciate having seen Dr. F. Laurendeau's letter to you. We are pleased to correct our oversight and to give credit to Dr. Laurendeau for his excellent article on this fascinating disease.

T. K. SCOBIE, MD, FRCS[C]

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Ottawa Civic Hospital,
Ottawa, Ont.

SURVIVAL IN CARCINOMA

To the Coeditors:

Dr. Chandler Smith (Interpreting the rate of survival in carcinoma. *Can J Surg* 18: 129, 1975) has discovered that treatment is not the only determinant of the survival of cancer patients. This scarcely justifies his preposterous plea to ignore the facts of what happens to cancer patients and base treatment on a "sound principle" of therapy. Though at one time it was acceptable to found treatment on hopes, dogmas, traditional beliefs and other sound principles, 20th century Canadian surgeons will prefer the scientific method of prospective, randomized controlled clinical trials, recognizing their limitations. Even though Dr. Smith has trebled the length of his otherwise identical statements of 1970,[1] 1971[2] and 1972[3] by repetitions, it is still absurd to recommend that treatment rest on anything but patient outcome.

J. E. DEVITT, MD, FRCS[C]

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To the Coeditors:

In reply to Dr. Devitt's letter, the main determinant of survival is the spread of the tumour. Tumour spread is estimated by clinical staging, which is extremely inaccurate. The number of patients who can be cured by one method but not the other is extremely small. There is, accordingly, no way to tell from a given result whether it is due to a favourable method of treatment or a fortuitous staging of the disease. This difficulty is obviated by realizing that the rate of survival does not express the quality of the method, it expresses the proportion of patients with localized disease. It is, therefore, more reliable to select the method by compliance with the principle of treatment than by comparison of the rates of survival.

C. SMITH, MD

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Kansas City General Hospital and Medical Center,
Kansas City, Missouri.

BOOK REVIEWS

MALIGNANT LYMPHOMA: NODAL AND EXTRANODAL DISEASES. Yeu-Tsu Lee and John S. Spratt, Jr. 411 pp. Illust. Grune & Stratton Inc., New York; Longman Canada Limited, Toronto, 1974. $20.25.

During the past five years the role of the surgeon in the management of patients with malignant lymphomas has greatly increased. This new role in oncology is recognized in the publication of this monograph about lymphomas by two surgeons.

The authors have assembled an extensive review of recent literature on lymphomas, concentrating particularly on published reports of series of patients. Not unexpectedly, they concentrate on the role of the surgeon in diagnosis, staging laparotomies, and the management of complications of both the disease and the therapy, particularly radiotherapy.

Included in the book is the authors' own experience in the investigation and treatment of 527 patients with malignant lymphomas seen at Ellis Fischell State Cancer Hospital, Columbia, Missouri, from 1940 to 1971.

Perhaps the most important chapter is the one devoted to the technique of staging laparotomy and splenectomy with an assessment of current knowledge of the value of, and the indications for, the procedure. The cumulative experience from 22 institutions covering over 1000 patients who have undergone laparotomy resulted in alteration of the stages of an average of 24% of patients with Hodgkin's disease to a more-advanced stage and 15% to a less-advanced stage after staging laparotomy. The procedure had a mortality rate of 0.5% with a postoperative complication rate of up to 30%. The authors appear to favour routine laparotomy for almost all patients with Hodgkin's disease and possibly for patients with non-Hodgkin's lymphomas also, but they concede that it remains to be shown whether laparotomy will alter the prognosis of these diseases.

The sections devoted to the economics of radiotherapy and the radiobiology are too brief to be of great value.

All physicians and surgeons involved in the investigation and management of patients with these diseases will find this book a valuable review of published experience of the last decade.

R. HASSELBACK

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The Princess Margaret Hospital,
Toronto, Ont.

MECHANICAL DISORDERS OF THE LOW BACK. H. F. Farfan. 247 pp. Illust. Lea & Febiger, Philadelphia; The Macmillan Company of Canada Limited, Toronto, 1973. $19.25.

Chemonucleolysis and denervation of the facet joint by high-frequency electroprobe for the treatment of various back disorders have brought about many new concepts of the etiology of low back pain. Therefore, a basic understanding of the intervertebral disc and its related structures is essential.

This book is a tribute to Dr. Farfan's years of clinical and laboratory research on the mechanical aspects of low back pain. The author discusses the embryology, anatomy and biomechanics of the lumbar spine in detail. The basic concept of the spine as an integral arrangement of many complex biologic and mechanical units is well formulated. The chapters on the movements of the lumbar spine and the degeneration of the intervertebral joint are exceptional and should be read by all who are interested in the subject.

However, the complexity of many of the chapters—especially those on torsion, hydraulics, stress analysis, and muscle action—limit the practical usefulness of this book. These chapters contain too much theory and mathematics to be of practical value to the average physician. Many of the concepts are based on years of detailed analysis and direct research and are illustrated by extremely complex charts and graphs.

The author did not write this book for the novice. It is written for the physician whose primary interest is low back pain, who is well-read in the pathology of the back and whose clinical expertise and acumen in diagnosis need supplement; for him this book would indeed be an addition to his intellectual armamentarium.

I. MACNAB

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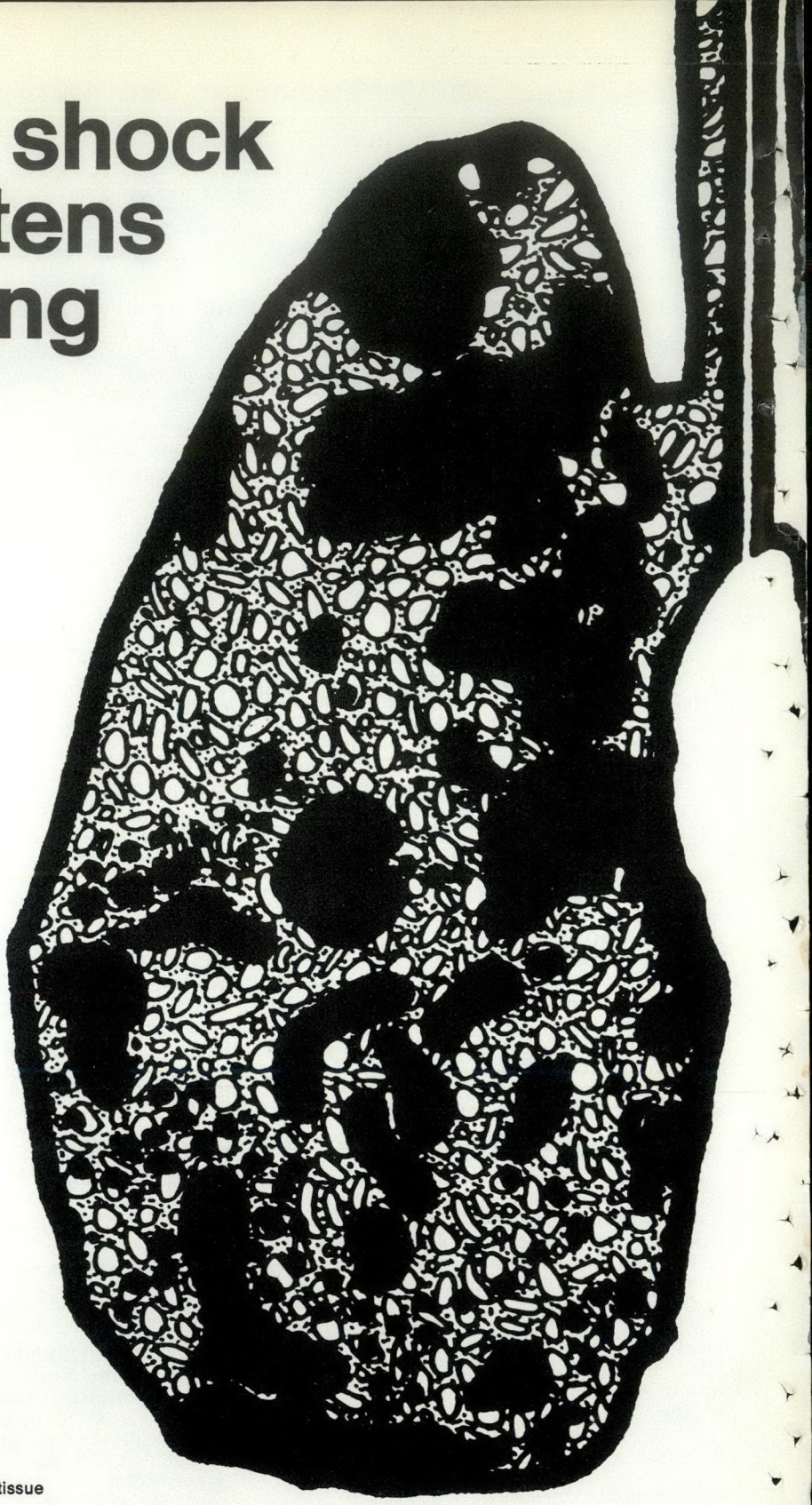
OPERATING THEATRE TECHNIQUE. A Textbook for Nurses, Technicians, Operating Department Assistants, Medical Students, House Surgeons and Others Associated with the Operating Theatre. Raymond J. Bridgen. 698 pp. Illust. Churchill Livingstone, Edinburgh; Longman Canada Limited, Toronto, 1974. $45.00.

The author of this book discusses, briefly, all

when shock threatens the lung



Abstract visualization of lung tissue

Solu-Medrol

helps reduce pulmonary damage and increase survival rates

- preserves lysosome and cell membranes, thereby preventing the release of destructive lysosomal enzymes[3]
- preserves platelets thereby reducing the risk of intravascular coagulation[1]
- preserves leukocyte integrity thereby helping to maintain the pulmonary architecture[1]

The recovery of patients in shock is often complicated by a pattern of deteriorating pulmonary function. This pulmonary insufficiency progresses despite restoration of hæmodynamic balance and apparent stabilization of the acute episode.

Under conditions of prolonged shock, lack of oxygen at the cellular level causes alterations in the oxygen-carbon dioxide exchange mechanism. These changes in cell metabolism lead ultimately to interstitial œdema and perivascular hæmorrhage.[1] Polymorphonuclear leukocytes aggregate in the pulmonary capillaries and obstruct the pulmonary vascular bed. As these trapped cells break down, they release lysosomes, tiny subcellular particles containing proteolytic enzymes.[1] These enzymes attack their host cell and go on to damage or destroy other cells.[2] The resulting tissue damage may not readily repair itself even if the shock patient survives.

When administered in conjunction with standard therapeutic measures, Solu-Medrol exerts a protective effect on the lung and improves the patient's chance of survival.

Prescribing information on following page

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2. Janoff, A. (1964). Shock, p. 93.
3. DeDuve, C. (1964). Injury, Inflammation and Immunity, p. 283.

aspects of management of an operating room, including operating room committees, training required by nursing personnel and suggested guidelines for job descriptions for all levels of staff in the operating room. He covers the physical aspects of an operating room: lighting, furniture and equipment, as well as static electricity, suture materials, "prepping", "draping" and storage and handling of supplies and equipment (e.g. monitors and anesthetic equipment). There is a detailed chapter describing methods of sterilizing and how the various methods destroy bacteria.

Bridgen then discusses basic instruments and their use for all types of surgery, and provides a chapter on techniques for use and application of plaster casts. He does not attempt to include many of the newest advances in surgery, but he does discuss up-to-date methods and equipment for all areas surrounding surgery—anesthesia, sterilization, ventilation.

This book is based on operating theatre practices in Great Britain, and there is perhaps confusion in the terms used to describe some equipment and procedures. However, it is well written, easy to read and clearly illustrated. It contains valuable information which should be known by all nursing staff who work in operating rooms. It could be an excellent reference for education programs for new nursing staff, and still it is interesting reading for the experienced operating room nurse, for medical students, interns and residents.

In summary, the book gives an excellent overall view of basic operating room knowledge.

E. A. HUNTER

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SPINAL DEFORMITY IN NEUROLOGICAL AND MUSCULAR DISORDERS. Edited by James H. Hardy. 262 pp. Illust. The C. V. Mosby Company, St. Louis; The C. V. Mosby Company Ltd., Toronto, 1974. $31.00.

Surgeons who treat spinal deformities soon become aware of a group of patients with scoliosis who are different from the majority. The cause, the prognosis, the response to bracing and the response to surgery of their spinal deformities are all different. These patients with relatively rare neurologic or muscle diseases present problems that tax the wisdom and skill of even the most experienced surgeons.

This book, therefore, is welcome and will be widely and eagerly read. It brings together the wisdom of nine orthopedic surgeons, three neurologists and one anesthetist on the diagnosis, natural history and management of spinal deformity in neuromuscular disease.

Sections on neurologic evaluation, spinoce-

rebellar degenerative diseases and motor unit disorders present, briefly, reference material for differentiating various neuromuscular diagnostic problems. The section on muscular dystrophy offers an original survey of the problem of spinal deformity in that disease, which should stimulate orthopedic surgeons to recognize their responsibility in this rather neglected area, and provides rational suggestions for management. There are also mature summaries of current views on the management of spinal deformity in poliomyelitis, spinal dysraphism, and congenital spinal deformities, and of the scoliosis associated with cerebral palsy, neurofibromatosis and trauma. The book is completed by a chapter on the special considerations influencing anesthesia for these patients when they undergo spinal operations.

Much has been written on scoliosis but little on the material covered in this book. Orthopedic surgeons interested in spinal surgery should read this small volume and use it as a foundation from which to progress in their own practice and thinking on this subject.

D. A. GIBSON

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SURGICAL ASPECTS OF HAEMODIALYSIS. P. R. F. Bell and K. C. Calman. 130 pp. Illust. Churchill Livingstone, Edinburgh; Longman Canada Limited, Toronto, 1974. $16.25.

In a text of approximately 120 pages Bell and Calman provide a good account of ways to obtain access to the circulation in patients requiring hemodialysis. The literature is well documented, there are clear line drawings of surgical procedures and throughout there is a balanced account of individual procedures and the part each may play in a planned approach to surgical access in individual patients, including dialysis in children. Over 20 surgical procedures are mentioned in this text, but one or two are omitted; for example, the single-needle technique, the Buselmeier shunt, the Sparks-mandril internal prosthesis and the modified bovine arteriovenous anastomosis. No mention is made of whether intermittent distension of a new arteriovenous fistula by a blood pressure cuff expedites maturity of the fistula; nor is there mention of the possible role of communications between the superficial and deep venous system, especially in limiting the use of arteriovenous fistula in the leg. The authors avoid the question of who should look after vascular access in chronic renal failure programs, specifically whether this should be done by several surgeons, a single surgeon or his resident. In spite of these omissions this is a valuable description of the present state of the art and should be on the bookshelf in every dialysis unit and of every surgeon concerned with hemodialysis.

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Edmonton, Alta.

SURGICAL DISEASES OF THE CHEST. 3rd ed. Edited by Brian Blades. 818 pp. Illust. The C. V. Mosby Company, St. Louis; the C. V. Mosby Company Ltd., Toronto, 1974. $44.65.

The third edition of Blades' text contains all the disadvantages that one would expect in a book containing contributions from a number of authors in that it has personal bias and irregularity of presentation from chapter to chapter. Some chapters—those on basic physiology, cardiac catheterization and heart valve replacement are examples—are clear and easy to read, but others are difficult. The 25-page chapter devoted to the surgery of tuberculosis seems excessively long. The chapter on extracorporeal circulation suggests a flow rate infinitely lower than anybody in current practice would use. The pacemaker chapter is reasonably good; it is gratifying to see pacemaker follow-up clinics advocated so that premature replacement of expensive packs can be avoided.

Although the illustrations on the whole are good, the drawings of esophageal atresia are reversed and the diagram for the surgical approach to a tracheoesophageal fistula is completely incorrect. The chapter on ancillary procedures is almost too brief to be of value. One would have liked more details on the management of problems in the postoperative patient, particularly concerning patients in the intensive care unit. The references are certainly not current; one chapter containing 300 references had only 12 that referred to work published later than 1970. The book is well printed but at a price of $45.00 I find its purchase difficult to recommend.

R. B. LYNN

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SYMPOSIUM ON NEOPLASTIC AND RECONSTRUCTIVE PROBLEMS OF THE FEMALE BREAST. Volume VII. Edited by Reuven K. Snyderman. 120 pp. Illust. The C. V. Mosby Company, St. Louis; The C. V. Mosby Company Ltd., Toronto, 1973. $18.40.

The publication of this symposium on neoplastic and reconstructive problems of the female breast is timely in the light of rethinking by the medical profession regarding this neoplasm. Its value will be increased in the United States by the coincidental occurrence of mam-

mary neoplasms in prominent political families.

The first chapter succinctly describes the magnitude of the breast cancer problem. This is followed by a good discussion and description of premalignant lesions. The use of subcutaneous mastectomy as the treatment of choice for minimal breast cancer is presented to stimulate surgeons to rethink their attitudes regarding radical mastectomies.

The chapter on breast augmentation would be more suited to a question and answer article in a women's magazine and is not appropriate for a medical publication. Unfortunately, this question and answer motif continues throughout most of the book, which falls somewhere between a lay and scientific publication.

The illustrations and diagrams are few. The book provides little new information and is not complete enough for the uninitiated doctor who has to deal with cancer and reconstruction of the female breast.

W. R. WATERS

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Winnipeg, Man.

SYMPOSIUM ON RECONSTRUCTION OF THE AURICLE. Volume 10. Proceedings of the Symposium of the Educational Foundation of the American Society of Plastic and Reconstructive Surgeons, Inc., held at Charlottesville, Virginia, June 28-29, 1973. Edited by Radford C. Tanzer and Milton T. Edgerton. 312 pp. Illust. The C. V. Mosby Company, St. Louis, 1974. $44.65.

The educational foundation of the American Society of Plastic and Reconstructive Surgeons continues to hold symposia on specialized subjects. The proceedings are published in full but without any attempt to produce a cohesive book. In spite of this the symposia are usually presented by international experts, and this volume is no exception.

The first part of the book is devoted to the anatomy, embryology and classification of ear deformities. There is an excellent definitive chapter on the growth of normal and reconstructed auricles by Farkas. Many of the "experts" challenge his data throughout the book but none can support their "observations" by scientific recording.

The section on reconstruction for microtia is an excellent collection of the methods available with explicit descriptions by Tanzer using cartilage and Cronin using Silastic. But little space was given to Brent, who has produced one of the simplest methods of carving cartilage for the reconstruction of the microtic or deformed ear. There are sections on reconstruction in lesser ear deformities, excluding the outstanding ear and traumatic deformities. One brief section considers the management of associated hearing and middle-ear problems.

The last part of the book concerns associated congenital facial deformities. This is too brief to be explicit or to be considered in depth. The chapters on the use of osteotomies to correct skeletal deformities seem to have been used to increase the appeal of the book and are too superficial to be of much value. Photographs showing correction of hemifacial microsomia 1 month after surgery when the face is still swollen are of no value.

In general, this is a useful book for those concerned with ear reconstruction. It is marred by considerable repetition by the different authors on the same subject. However, almost every known method of reconstruction is described, though where only line drawings are used, the inexperienced surgeon has no way of evaluating the results. Probably the best summary is provided by Curtin acting as devil's advocate. "Not everybody should do ear reconstructions. Also many if not most cases should be sent to multiprofessional craniofacial teams to evaluate the timing of the multiple procedures if there are associated deformities."

I. R. MUNRO

Division of plastic surgery,
Sunnybrook Medical Centre,
Toronto, Ont.

THE YEAR BOOK OF ORTHOPEDICS AND TRAUMATIC SURGERY 1974. Edited by H. Herman Young. 478 pp. Illust. Year Book Medical Publishers, Inc., Chicago, 1974. $19.00.

In this volume of abstracts (now in its 74th year of publication) in the orthopedic, traumatic surgery field, over 300 pertinent articles are reviewed in detail. In addition, most of the abstracts contain a personal note at the end by one of the reviewing editors. This excellent little book reviews the world literature and includes a large variety of medical journals, not only orthopedic but many others, in the collection.

This book is highly recommended for all hospital and medical school libraries, as well as for all practising orthopedic surgeons.

E. C. PERCY

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Montréal, Qué.

INDEX TO ADVERTISERS

Books Received

Common Duct Stones. Frank Glenn. 135 pp. Illust. Charles C Thomas, Publisher, Springfield, Ill., 1975. $11.50.

Current Management of Trauma in Surgery and General Practice. Edited by Teruo Matsumoto. 382 pp. Illust. Charles C Thomas, Publisher, Springfield, Ill., 1975. $23.50.

Foundations of Surgical Nursing. Jane Forrest. 92 pp. Illust. Edward Arnold (Publishers) Ltd., London; The Macmillan Company of Canada Limited, Toronto, 1974. $3.95. Paperbound.

Clinical Thyroidology. Joel I. Hamburger. 206 pp. Illust. Joel I. Hamburger, Northland Laboratory, P.C., 20905 Greenfield, Ste. 300, Southfield, Michigan 48075, USA, 1974. $9.75. Paperbound.

Hypothermia in Biology and in Medicine. Vojin Popovic and Pava Popovic. 305 pp. Illust. Grune & Stratton, Inc., New York; Longman Canada Limited, Toronto, 1974. $26.00.

Orthopaedic Surgery in Infancy and Childhood. 4th ed. Albert Barnett Ferguson, Jr. 791 pp. Illust. The Williams & Wilkins Company, Baltimore; Burns & MacEachern, Toronto, 1975. $53.50.

Réanimation et Médecine d'Urgence 1974. Edited by M. Goulon, M. Rapin, A. Barois and F. Nouailhat. 365 pp. Illust. Expansion Scientifique Française, Paris, 1974. Paperbound.

Small Fragment Set Manual. Technique Recommended by the ASIF-Group. Urs Heim and Karl M. Pfeiffer in collaboration with H. Ch. Meuli. Translated by R. Kirschbaum and R. L. Batten. 299 pp. Illust. Springer-Verlag, New York, 1974. $54.20.

The Toronto General Hospital 1819-1965: A Chronicle. W. G. Cosbie. 373 pp. Illust. The Macmillan Company of Canada Limited, Toronto, 1975. $9.95.

The Year Book of Plastic and Reconstructive Surgery 1975. Editor: Frederick J. McCoy, Associate Editors: Reed O. Dingman, John C. Gaisford, B. W. Haynes, Jr. and others. 351 pp. Illust. Year Book Medical Publishers, Inc., Chicago, 1975. $25.00.

NOTICE

CANCER SYMPOSIUM

The Third International Symposium on Detection and Prevention of Cancer is to be held in New York City at the Americana Hotel, April 26 to May 1, 1976. For further details contact: Dr. H. E. Nieburgs, Mount Sinai School of Medicine of the City University of New York, Fifth Ave. and 100th St., New York, NY 10029, USA.